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GUEST EDITORIAL

The Opportunity for Study and Investigation in Practice

WE PHYSICIANS are fortunate in that we have the ever present opportunity to learn, and are constantly challenged by the fact that there is much new presented to us every year with which we must make ourselves familiar, as well as much old that we must review. What appear to be fundamental concepts of physiological processes today may be changed tomorrow because of new discoveries or re-evaluation of these concepts.

It is this challenge that keeps our interest stimulated so that our work need never become dull. Physicians in every specialty have this opportunity, but that of the general practitioner seems to be the greatest since he has a broader field to cover. He can always find something new and fascinating to discover for himself each day and thus maintain his enthusiasm. He can review what he once learned and often in doing so find something entirely new.

The opportunity to do scientific medicine is not lost in practice. As students and house physicians we were given all types of patients to examine, study and treat. The signs and symptoms, pathological physiology, pharmacology and therapy were reviewed, and the patient's emotional reaction to his disease was considered. Many laboratory facilities were at our disposal, consultants were available in all specialties, and the diagnosis often became obvious as the different links in the diagnostic chain were fitted together to make a clinical syndrome with which we were familiar.

The physician in practice has the same problems, but often with fewer facilities and without available consultation. This does not make the problems less intriguing, but more so, and he can often arrive at a correct diagnosis alone or with the help of minimal facilities if he observes carefully and draws thoughtful conclusion from his observations. The pleasure thus afforded is worth many hours of thought and work. Each patient therefore presents an opportunity to do scientific medicine since science is not a matter of gadgets and special equipment, but more often a matter of observation, recording and evaluation.

We sometimes hear young men bemoaning the lack of opportunity to continue with clinical investigation because of the necessity of going into practice. It is true that one cannot select the type of problems presented in practice, nor can all of the facets of the investigation be controlled; however, each patient that is seen in practice is a problem in clinical investigation. Pathological physiology in all of its manifestations is encountered and can be studied. One can make observations, can accumulate scientific data and make contributions to medical literature by studying the patients seen

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in practice. He can review the normal and abnormal physiology of his patients, can compare his work and observations with those of his colleagues in large centers and often finds himself happier as a result of this comparison. He need not lose his investigative interest or perspective, nor need he fall far behind these colleagues in his knowledge. This does require extra work and reading, but the satisfaction gained is worth the effort.

The infectious enthusiasm of our medical school teachers which we absorb is often lost because it is not kept alive after we leave their association. An academic atmosphere depends on the individuals who create it, not on the building in which it is created. Therefore, each physician can create his own academic atmosphere within himself by study and by association as much as possible with others who make the same effort. This, too, is infectious and will spread to other physicians, helping to create in them the enthusiasm for study, thus allowing them more pleasure from their work.

It is not easy, we are sure, for the physician in practice always to do what he wishes to do, because the pressure of work often keeps him from reading and study, but if he continually makes an effort to reserve a little time each day for this purpose and to consider each patient an intellectual challenge, he will obtain much satisfaction and maintain a constant enthusiasm for his work.

It has been said that the study of medicine is a plea ure, but the practice is a chore. This is probably true if practice is done without study.

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Floral Eponym

MACADAMIA

MACADAM, JOHN, 1827-1865

John Macadam. M.D. Secretary of the Victoria Philosophical Institute, studied medicine at the University of Glasgow and chemistry at Edinburgh. He was a member of the legislative Assembly of Victoria (1859-64) and Postmaster General (1861-2). He died at sea while returning to New Zealand.

Macadamia is a genus of five species of slow growing Australian trees . M. ternifolia is known as the Queensland nut.

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DON'T DO THIS FOR ASTHMA*

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In 1932, "An Analysis of the Work of the First Year of the Allergy Clinic of the University of Virginia Medical School"[1] was read before the Annual Meeting of this Society. In the introduction to that report, one of the comments was, "The average physician should be able to diagnose and treat the allergic diseases with the same facility and confidence with which he diagnoses and treats other diseases of the cardio-respiratory, genito-urinary, gastro-intestinal, neuro-muscular, and muco-cutaneous systems." In the twenty years since then there have been perhaps 100,000 allergy patient visits, 39,977 in 1946-1950 inclusive. A large percentage of these visits were made by asthma victims. In studying these cases many therapeutic errors have been observed. Tuft[2] has recorded similar observations. This extensive experience has led to the reluctant conclusion that the average physician of today manages asthma far less effectively than it was hoped he would twenty years ago. An analysis of and recommendations for the correction of some of the more commonly observed errors in the management of asthma forms the basis of this anniversary report from the University of Virginia Allergy Clinic. To emphasize these errors, fifty-odd "DON'TS" are discussed dogmatically under the headings: symptomatic treatment, treatment of infection, elimination diets, information obtained from skin tests, injection of allergens, classification of asthma, and miscellaneous.

SYMPTOMATIC TREATMENT

In asthma the patient wheezes and is short of breath because his airways are narrowed. The narrowing is due to increased secretions, swelling of the mucosa and submucosa, and to spasm of the bronchial muscles. The relative importance of these three factors varies from patient to patient and from time to time in the same patient. Drugs most effective in correcting these factors are: the sympathomimetics, xanthines, expectorants, sedatives, and, more recently, ACTH and cortisone.

THE SYMPATHOMIMETICS relieve asthma because they dilate the bronchi by direct stimulation of the bronchodilator muscles, absorb exudate and reduce swelling, and increase vital capacity.

Don't Over-Dose with Aqeous Adrenalin. 0.2 cc. of 1:1000 adrenalin (epinephrine) usually acts quickly and effectively for several hours. Repeat 2 or 3 times at 10-minute intervals if necessary. Additional or larger doses are not apt to help when enough has been given to speed the pulse perceptibly, or to cause tremor, palpitation, apprehension, or pallor. It seldom causes troublesome side effects when just enough is given to relieve the attack. Most supposed instances of intolerance are due to all-too-common over-doses of 0.5 to 1.0 cc.

Don't Hesitate to Teach Asthmatics to Give Themselves Adrenalin. The knowledge that prompt relief is at hand removes much of the fear which so often converts a mild attack into a severe one.

Don't Use Adrenalin in Oil to Relieve an Acute Attack. It takes effect slowly as a rule. Its proper use is to maintain relief gotten from aqueous adrenalin or other remedies and to abort regularly recurring attacks.

Don't Give Adrenalin in Oil Until It Has Been Thoroughly Emulsified by warming in the hand and shaking vigorously for one to two minutes. One ampoule of adrenalin in oil contains the equivalent of 2.0 cc. or 10 normal doses of aqueous adrenalin. Unless it is thoroughly emulsified, severe adrenalin over-dose reactions are to expected.

EPHEDRINE

Ephedrine acts more slowly and less effectively than adrenalin. It is effective by mouth in doses of 25 to 50 mg. It may cause wakefulness at night and nervousness during the day. Its chief uses are to relieve mild attacks and chronic mild wheezing and to abort recurring attacks. Many good combinations of ephedrine, barbiturates, xanthines, and one with potassium iodide are on the market.

Don't Expect Ephedrine to Relieve Severe Attacks. Don't Expect Adrenalin 1:100, Isuprel 1:200,

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Read before the Annual Meeting of The Medical Society of Virginia at Richmond, September 28-October 1, 1952.

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Vaponephrine, or other nebulized preparations to relieve severe attacks. Their effectiveness is comparable to but is quicker and more transient than ephedrine by mouth.

Don't Let Patients "Chain Smoke" Their Nebulizers.

Don't Use Substitutes for Adrenalin and Ephedrine Routinely.* They are not as effective as a rule, but they can be tried in the occasional case when adrenalin and ephedrine are not well tolerated.

XANTHINES relieve asthma by relaxing constricted bronchi, stimulating the respiratory center and increasing vital capacity. Aminophylline is the most widely used one. Intravenously it is the most effective remedy for troublesome attacks of asthma which are resistant to adrenalin.

Don't Expect Aminophylline by Mouth or By Rectum to Relieve Severe Attacks. These routes are effective for mild attacks and for prophylaxis. Gastric or rectal irritation sometimes prevent their continued use.

Don't Use Intramuscular Aminophylline, in spite of the label on the ampoules. It is cruelly painful as a rule. The 2.0 cc., 0.5 gm. "intramuscular" ampoules are used intravenously, without dilution, in this clinic. They are cheaper and handier to use than the 10 to 20 cc. sizes.

Don't Give Aminophylline Intravenously Rapidly. 0.25-0.5 gm. intravenously should take 3 to 5 minutes. A 25-26 gauge needle lessens the tendency to painful extravasations. Stop the injection immediately if the patient complains of anything except nausea, a queer taste, or flushing. Nausea helps to relieve asthma frequently. Truly untoward reactions are rare, although several deaths have been reported after intravenous aminophylline given for relief of cardiac asthma due to acute coronary insufficiency.

Other xanthine preparations are effective, but are less widely distributed. If aminophylline causes reactions, caffeine sodium benzoate can be substituted by mouth or injection, quite satisfactorily.

EXPECTORANTS.

POTASSIUM IODIDE is the most widely used, the oldest, and perhaps the most effective expectorant for relief of asthma. It is effective in keeping sputum fluid, but it doesn't seem to liquefy sputum which is already thick and tenacious. Occasionally, no other remedy is needed.

Don't Prescribe Potassium Iodide Unless Direct Questioning Discloses No Troublesome Reactions To It.

Don't Use Compound Tincture of Benzoin, Camphor, Menthol Inhalation. Plain steam will do as well and does not cause the troublesome reactions sometimes caused by strong smelling drugs.

Many allergists concoct effective expectorant formulae from ipecac, ammonium chloride, ether, chloroform, guaiacols, volatile oils, and citrates.

SEDATIVES.

Don't Use Morphine Or Its Derivatives. They are sensitizers, respiratory depressants, anti-expectorants, habit-formers, often lethal, and are unnecessary.

BARBITURATES may be helpful in three ways: by counteracting the stimulating effects of sympathomimetics and xanthines, by reducing the hyperexcitability of the respiratory reflexes, and by relaxing the smooth muscles of the bronchi. They are widely used and are usually well tolerated. CHLORAL HYDRATE is a frequent substitute. DEMEROL, though less desirable, may be helpful, too. But all three drugs may sensitize, depress respiration, and lead to addiction.

Don't Give Any Sedative Without Being Sure It Has Not Caused Asthma, Mental Aberration, Respiratory Depression, Or Other Undesirable Reactions Previously. Respiratory depression, which many sedatives cause, must be avoided in the tired asthmatic.

PARALDEHYDE should be used more often. It does not sensitize nor depress respiration in pharmacological doses.

OXYGEN. *Don't Use Oxygen Routinely.*[3] Oxygen rarely relieves asthma. It does nothing to reduce swelling, secretion, or bronchial spasm. On the contrary, oxygen is a potent antiexpectorant. It is probably responsible for the troublesome inspissation of sputum in many cases of status asthmaticus. Most asthmatics oxygenate their blood and blow off carbon dioxide adequately. Oxygen may occasionally reduce the respiratory effort sufficiently to cause a dangerous retention of carbon dioxide. In the cyanotic asthmatic, oxygen may be used cautiously and only as long as he is cyanotic. It is best given

*Orthoxine, Isuprel, racephedrine, benzylephedrine, propadrine, neosynephrine, butanephrine.

by intermittent demand positive pressure to enhance expiratory loss of carbon dioxide. The asthmatic who is receiving oxygen should be under constant observation. Oxygen should be stopped promptly if cyanosis deepens, if mental acuity lessens, or if respiration becomes shallower or slower.

The oxygen tent without oxygen but with filtered, cooled, and moistened air protects the patient from many inhalant allergens, is not dangerous, and fulfills the demands for dramatic therapeutic ritual.

The chief reasons for not using oxygen routinely are: it doesn't help the asthma and, by diverting the physician's attention, its use tends to deprive the patient of effective remedies. He survives the attack in spite of and not because of oxygen. There are exceptions, of course.

ACTH and CORTISONE are symptomatic remedies. They do not cure, nor is there evidence that they will prevent late complications of chronic asthma.

Don't Use ACTH or Cortisone Except: (1) to relieve status asthmaticus; (2) when prolonged treatment, based on comprehensive and repeated analyses of the patient's asthma problems, has failed; (3) to supplement unsuccessfully treated seasonal pollen asthma.

Status asthmaticus means asthma which has not been relieved by adrenalin, aminophylline, potassium iodide, sedatives, and antibiotics in pharmacological doses. 25 mg. ACTH intramuscularly every 4 hours improves status asthmaticus within 4 to 20 hours usually. An initial slow I.V. drip of 10-15 mg. in 1000 cc. of 5% glucose seems to bring quicker relief at times. Reduce the dose and lengthen the interval between doses, rapidly, as soon as the attack subsides. Then stop ACTH.

Cortone usually takes several hours longer than ACTH to relieve severe asthma. Otherwise it seems as effective. It works faster by mouth than injection, a real advantage. An initial oral dose of 100 mg., then 75 to 50 mg. every 4 to 6 hours, brings relief in 12 to 30 hours in all but the most severe attacks. The dose should be lowered and the interval lengthened, as with ACTH, as soon as the attack subsides.

Oral cortisone is preferred as maintenance therapy in chronic asthma resistant to other therapy. After relief is obtained use the smallest ration which will keep the patient comfortable.

ANTIHISTAMINES are apt to be poor anti-asthmatic remedies.

Don't Use Antihistamines Until Sympathomimetics, Xanthines, Sedatives, and Expectorants Have Failed. Antihistamines may further inspissate sputum. They can be used for their sedative and adrenalin-sparing effects to supplement the sympathomimetics and xanthines. They seem to be more effective in children than in adults.

TREATMENT OF INFECTION

Infection alone or complicating atopy is a common cause of severe asthma. Infection should be assumed to be present when the patient coughs himself into an attack of asthma and there is pus in the sputum or in the nasal discharges; when the nasal and pharyngeal mucosae are red; when the uvula is red and swollen or wrinkled; when there is fever or leucocytosis; or when there is evidence of sinus disease. Acute infectious asthma responds to specific antibacterial therapy almost as promptly as other infections do. Asthma associated with chronic infection responds favorably but less dramatically.

AEROSOL THERAPY, using any standard nebulizer and any convenient source of clean compressed air or oxygen, is frequently an effective adjunct in controlling bronchial infection. In this clinic 25,000 units of penicillin and 25 milligrams of streptomycin in 1 cc. of either 1:10,000 adrenalin, 1:1000 isuprel, or 0.1% neosynephrine is nebulized every 2 to 3 hours to supplement other antibacterial therapy. Bacitracin, polymyxin, sodium caprylate and other antibiotics have been used when indicated by culture and sensitivity tests.

Several precautions are necessary for the successful treatment of infectious asthma.

Don't Stop an Effective Antibacterial Agent Until the Episode Being Treated Is Over. Failure to continue the effective agent for from five to ten days often leads to mild chronic infectious asthma.

Don't Continue One Antibacterial Agent Unless It Produces Striking Benefits within three or four days and disappearance of pus within seven to ten days. Change to another.

Don't Forget That Suppression of One Type of Organism May Promote Growth of Other Organisms, particularly pyocyaneus, aerogenes and proteus, and occasionally monilia. Therefore, repeated cultures are necessary when infection persists or flares up

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during treatment. The antibacterial agents must be changed with the changing flora.

Don't Fail to Seek Help from the Rhinologist in Infectious Asthma. He is an indispensable partner in its treatment. He should lavage antra, aspirate the other sinuses, remove polyps, straighten obstructing nasal septa, employ X-ray therapy and other conservative but vigorous treatment. Radical sinus surgery is still necessary occasionally.

Don't Ignore Nasal Polyps. They should be removed as they form in order to eliminate the role of the naso-bronchial reflex in the production of asthma. They are seldom seen except in the presence of allergy plus infection.

ELIMINATION DIETS

Elimination diets are essential to the management of asthma. But several precautions are necessary to prevent their misuse.

Don't Tell Patients They Are Allergic to a Food Unless Avoidance of It Is Beneficial and Eating It Causes Aggravation of Asthma.

Don't Tell a Patient He Is Allergic to a Food Because He Has a Positive Skin Test to It. Positive skin tests are discussed later.

Don't Eliminate a Food from a Patient's Diet for Longer Than a Specified Diagnostic Period of three or four weeks unless the asthma is better when the food is avoided and worse when it is eaten. In this case, the food should be avoided indefinitely.

Don't Advise the Reduction of Intake of a Suspected Food in a Preliminary Elimination Diet. Instead, all traces of foods suspected by history or skin test should be avoided for a trial period of three or four weeks. The success of elimination diets depends largely upon the thoroughness with which the patient is told how to avoid suspect foods in disguised form. For example, the physician should remind the patient that mustard may be found in pickles, commercial mayonnaise, canned soups, and baked beans; egg white may be found on baker's rolls; milk in baker's loaf bread; wheat in gravy, fried chicken, spaghetti, macaroni, noodles, and creamed soups.

Don't Hesitate to Eliminate Milk or Any Other Food for a Trial Period, when suspected by history or skin test. No one food is essential. Milk is a very convenient food but is more a housekeeping and dietary habit than a necessity. By the same token:

Don't Fail to Prescribe a Balanced Diet from permitted foods if the elimination diet is to last longer than 2 or 3 weeks. Elimination diets are disconcerting more because they disturb food habits and fads than because they disturb nutrition.

INFORMATION OBTAINED FROM SKIN TESTS

Don't Assume That a Positive Skin Test Indicates Clinical Sensitivity to the reacting antigen. A positive skin test is simply an etiological lead. Before the doctor or the patient can assume a causative role from a reacting food or inhalant, the patient must improve when avoiding the antigen and get worse when in contact with it, and/or he must be improved by injections of it.

Skin tests, with some reservations, reflect immunological experiences. A positive skin test represents pre-clinical, post-clinical, sub-clinical, or clinical sensitivity. A positive tuberculin skin test indicates that there has been an immunological experience with tubercle bacilli. Clinical tuberculosis cannot be implied from the positive test alone. It serves as an indication for further diagnostic studies. The same can be said for positive skin reactions to dust, egg, ragweed, and other allergens.

Don't Think of Skin Tests and Asthma Diagnostic Studies As Synonomous. Skin testing is helpful only in finding the causes of atopic asthma. They are useless in determining other causes of wheezing discussed below.

INJECTION OF ALLERGENS

It is important and undeniable that allergen injections do relieve asthma frequently. The mechanism by which relief is obtained from allergen injections is not clear. Whether it be due to desensitization, hyposensitization, blocking antibody production, non-specific induction of the adaptation phase of the alarm reaction, or other mechanism is not germane to this discussion.

Don't Start Allergen Injections Until Food and Inhalant Avoidance and Treatment of Infection, When Present, Have Failed to control the asthma. A notable exception is that treatment of seasonal pollen cases should be started as soon as the diagnosis is made.

Don't Expect Prompt Relief from Injections. It may take months to get satisfactory results. Relief from a few days of co-seasonal treatment with pollen

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extracts is an exception to this rule.

Don't Inject Extracts Just Because the Skin Test Is Positive. In this clinic there have been hundreds of examples of improper prior treatment of: grass pollen asthma with ragweed extract and *vice versa;* simple dust asthma with ragweed extract (in spite of the fact that they were better during the ragweed season than at other times); simple infectious asthma with dust extracts. It should be obvious that extracts for injection therapy should be selected from allergens to which the patient is exposed when he has symptoms. The likelihood of relief seems greater when there is the history of asthma from as well as a positive skin reaction to the allergen selected for injection.

Don't Over-Treat. A troublesome local reaction or an exacerbation of asthma or of nasal symptoms soon after an injection usually means over-dosage. Subsequent doses should be reduced well below the reacting dose. In allergy therapy the thought is appropriate that if a little is good, less (not more) may be better.

Don't Stop the Injections Because There Are Reactions to Them. Instead, dilute the extracts decimally until they no longer react. Dilutions of 1:1,000,000, or less, are sometimes necessary. Extracts which cause symptoms in conventional doses are usually helpful when suitably diluted.

Don't Interrupt Injection Schedules for Intercurrent Asthma, Colds, Other Illnesses or Pregnancy. This is a common practice which serves no purpose.

Classification of Asthma

Don't Assume That a Given Patient's Asthma Is Due to a Single Cause. Asthma is a syndrome due to narrowing of the airways, which can be precipitated by a variety of causes. These causes may be classified, for purposes of simplification, into atopic (food, inhalants, and drugs), infectious, reflex (nasal polyps, thyroid nodules), physical (heat, cold, dampness, sunlight), psychogenic, emphysema, bronchial obstruction (cancer, foreign bodies, mediastinal tumors), acute left ventricular failure. Most cases are due primarily to atopy and/or infection. But in these cases, reflex, physical and psychogenic components usually contribute to the severity and frequency of attacks. At times these contributory components play major roles. Attacks of asthma associated with bronchial obstruction or acute left ventricular failure may be indistinguishable from those due to milk or feathers.

Proper management of the chronic asthmatic requires a search for multiple causes of wheezing in every case. This search calls for the skilled use of history and physical examination in all, and X-rays and skin tests in most cases. In others the search will require bronchograms, bronchoscopy, biopsies, cultures and antibacterial sensitivity tests, and studies of heart and lung functions.

Miscellaneous

Don't Omit Asthma from the Differential Diagnosis in any case of cough or dyspnea, especially if nocturnal, recurrent, and with symptom-free intervals.

Don't Discard Asthma As a Diagnosis because there are no asthmatic squeaks during symptom-free intervals. When absent during the usual maneuvers of the chest examination, they can be elicited frequently by auscultation during forced expiration.

Don't Advise Parents to "Let the Child Outgrow His Asthma." Many do, most of them don't. This vicious cliché has been responsible for the unnecessary crippling of thousands of young adults by chronic lung failure, a common aftermath of untreated childhood asthma.

Don't Have House Pets, Cut Flowers, Rugs, Unnecessary Upholstery, Pillows, Curtains or Other Dust-Collecting Materials; and *Don't Use Insecticides, Scented Powders, Perfumes, Paint, Floor Wax, or Other Irritating Odors Around Asthmatics.*

Don't Smoke Around Asthmatics, Especially Children.

Don't Pile Pillows Around an Asthmatic During an Attack. Many asthmatics are aggravated by feathers. Support his orthopneic position with a chair or other non-allergenic objects under the mattress.

Don't Sweep. Use the vacuum cleaner, when the asthmatic is not around.

Don't Depend on the Clinical Examination for the Exclusion of Sinus Infection, particularly in children. X-ray examination is essential for the diagnosis, recognition, and cure of sinus infection.

Don't Allow the Asthmatic to Become Dehydrated. The hard work of breathing causes excessive loss of fluid from his lungs and skin. Inspissation of sputum, hemoconcentration, electrolyte imbalance, and their sequelae, may result.

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Don't Be Satisfied with Good But Incomplete Relief from Symptomatic Remedies. They do not postpone nor prevent the development of chronic asthma and its complications of chronic sinusitis, fibrosis, emphysema, chronic bronchitis, and ultimate chronic lung failure. Symptomatic remedies should supplement but should not replace specific therapy.

Don't Allow an Asthmatic To Get Sick Enough To Threaten Life Without Putting Him Where He Can Be Bronchoscoped, if necessary. Aspiration of inspissated sputum is a life-saving procedure occasionally, less often since the advent of ACTH and cortisone. The indications for bronchial aspiration are simple. First, a severe attack of asthma resistant to conventional remedies. Second, one or more areas of the lungs which have markedly diminished breath sounds persisting for several hours.

Don't Forget That the Frequency and Severity of Attacks Is Influenced by Emotional Tension in Many Asthmatics. Psychogenic factors are of major importance in a significant percentage of cases.

Don't Procrastinate. A detailed search for the causes of wheezing should be started with or soon after the first mild attack. Failure to do so results in much unnecessary suffering, chronic respiratory crippling and eventual death. Proper management, if begun before there are complications and continued as long as there is any asthma, will maintain the average asthmatic in good health indefinitely.

CONCLUSIONS

Medical schools do not prepare their graduates to treat allergic diseases with the same facility with which they care for other disorders of the cardio-respiratory, genito-urinary, gastro-intestinal, neuro-muscular and muco-cutaneous systems. Recognition of and correction of this glaring curriculum deficiency has been and still is painfully slow. This is particularly true of asthma, one of the most common causes of chronic organic illness.

An extensive experience at the University of Virginia has disclosed many errors in the management of asthma. An analysis of and recommendations for the correction of some of the more common errors has led to the compilation of the fifty-odd "DON'TS" which are the subject of this twentieth anniversary report of the Allergy Clinic of the University of Virginia Medical School.

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Film Cleared for TV.

Three films produced by the Metropolitan Life Insurance Company recently were cleared for use on educational and public service television programs. "Once Upon a Time"—a 10-minute animated cartoon dealing with state and highway safety; "Proof of the Pudding"—a 10-minute dramatic presentation showing the essentials of good nutrition and the relation between diet and health; "Be Your Age"—an 11½-minute drama depicting middle-aged, over-weight "John's" recovery from a heart attack and his subsequent adjustment to living with a handicapped heart. The films are available to county medical societies who desire to co-sponsor these films on television. They may be secured from Metroplitan Life Insurance Company, 1 Madison Avenue, New York 10, N.Y.

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TREATMENT OF GROIN HERNIAS*

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The previous paper on the treatment of burns fits in well with this paper on hernias, since by treating these two conditions the embryo surgeon can learn the fundamentals of surgery. Management of infection and electrolyte disturbances is well exemplified by treating burns, and application of the fundamental principles of surgical technique and surgical anatomy are embodied in the operative treatment of inguinal hernia.

It is often observed that a student can be taught to be a good surgeon if he learns to operate properly on hernias. Too frequently, however, in the course of a surgical residency, herniorrhaphies are turned over to the younger men who are not closely supervised in proper operative correction of the defect. This practice sometimes results in poor training of the residents and unsatisfactory results from operation. Hernia is one of the commonest remediable conditions encountered in surgical practice, and the industrial aspects of this condition are obvious. State industrial commissions recognize the obligations of employer toward employee regarding hernia, and correction of this condition is fully compensable.

From the historical viewpoint, attempts to cure or control hernias have been made for centuries. Reduction by taxis, treatment of bubonocele, etc., are described in ancient writing. The efficacy of trusses is attested by numerous advertisements in periodicals and newspapers, but surgery has far more promise of cure of the defect and relief of the symptoms. The vogue for injection treatment of hernias seems to have passed almost completely, although it is noteworthy that Watson's textbook on "Hernia," published less than fifteen years ago, contained over seventy pages on the technique of treating groin hernias by injecting sclerosing solutions.

The old saying, "There is nothing new under the sun" probably applies to this discussion and yet reiteration of the principles of hernia surgery serves to keep standard opinions and modern advances in mind.

Treatment of groin hernias by operative repair affords the polished surgeon as well as the student the opportunity to exercise many cardinal principles of surgical technique. Galen said, "No great and perfect work is ever accomplished at a single effort, or receives its final polish from one instrument." Application of thorough knowledge of anatomy, gentle surgical technique, and restoration of the normal abdominal support are combined to accomplish a satisfactory result. In no operation in general surgery is relief of symptoms more gratifying than in a properly executed herniorrhaphy.

The most important factor in the success of hernia repair is to utilize the proper operation for the various types of groin hernias. Because of the complicated regional anatomy, it is just as important not to transpose muscle planes for simple hernias as it is to provide adequate support for complicated defects.

Three types of groin hernias are ordinarily seen—indirect, direct, and femoral. Medical students find differentiation difficult, but on anatomical grounds these are separate distinct types. Indirect hernias are found when the *processus vaginalis* fails to fuse, and classically there is a rather long sac accompanying the spermatic cord in the inguinal canal. This type is usually not complicated by serious muscular defects. In this connection it is of interest that the etiology of hernias is not completely understood. Watson states that "the principal cause of hernia is undoubtedly the existence of a congenital sac." On the other hand, direct hernias are characterized by a weakness in the abdominal wall through which abdominal contents tend to protrude. This type is the true "rupture" and is most often due to weakening of the muscles from strain or advancing years. Femoral hernia also is due to a weakness of the muscular wall but the sac descends along the femoral vessels and presents in the thigh. Differentiation of groin hernias by palpation is difficult, and careful attention should be paid to physical examination, using the index finger to invaginate the scrotal skin and feel the hernia through the external inguinal ring. Indirect (or oblique) hernias accompany the spermatic cord and often descend into the scrotum, while direct hernias ordinarily present through a large external ring and are felt through a weakness superior and lateral to the pubic tubercle.

*Read before the annual meeting of The Medical Society of Virginia at Richmond, September 28-October 1, 1952.

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At operation it is our feeling that the indirect variety is best handled in as simple a way as possible, by high ligation and removal of the sac and by a minimum of muscle transposition. In children, complete excision of the sac with high ligation is all that is needed for cure, and since no muscular defect is present, there is no need for reconstruction of the floor of the canal. In adults with uncomplicated indirect hernias, the important step is complete removal of the sac, and when the floor of the inguinal canal needs buttressing the internal ring should be snugged around the cord and a Ferguson or Bassini type of muscle reconstruction performed.

Cure of direct hernia involves a more extensive operation. Since, in this condition, there is in reality a rupture of the muscular support, accurate anatomical restoration of the muscular floor must be achieved. It has been clearly shown by Anson in anatomical studies that the transversalis fascia inserts into Cooper's ligament, rather than into Poupart's ligament as was usually assumed. Therefore, any attempt at reconstruction should reinforce this tissue plane by the McVay technique of Cooper's ligament repair. Inasmuch as femoral hernias also involve a weak wall, it is our feeling that this operation is the procedure of choice for all direct and femoral hernias. The reason for this is that reconstruction of the inguinal canal by suturing the muscles to Poupart's ligament leaves a potential weakness through which herniation can develop between Poupart's ligament and the superior pubic ramus. This weakness is eliminated by the Cooper's ligament type of repair. The inguinal approach to femoral hernias rather than the subinguinal route allows better exposure of the neck of the femoral hernia sac and enables one to carry out the Cooper's ligament repair. In using this operation, it is essential to expose the superior ramus of the pubis from the pubic tubercle laterally to the femoral vessels by separating the fibers of the transversalis fascia. Cooper's ligament is then visualized as a tough fibrous structure adherent to the bone. Conjoined tendon and transversalis fascia are then securely sutured to Cooper's ligament from the femoral vessels to the pubic tubercle. In this way a thoroughly strong, satisfactory floor of the inguinal region is restored and any weakness in Hesselbach's triangle is effectively overcome.

There are several other points regarding technique worth mentioning. We prefer a transverse incision low in the right lower quadrant rather than the usual oblique herniorrhaphy incision. The incision is placed in a natural skin crease and produces a good cosmetic result. In babies this incision is particularly nice. The skin can be closed in infants and children with a continuous subcuticular suture of fine plain catgut. This is then covered with collodion and the patient may be discharged at the earliest convenience. A watertight closure is thus provided and there are no skin sutures to be removed.

We employ cotton sutures throughout except for catgut in ligation of the peritoneal sac and except when the skin is closed with subcuticular catgut. Strong, pliable wounds are obtained by cotton technique.

Early ambulation of herniorrhaphy patients has been our practice. We feel that this encourages prompt restoration of normal bowel and bladder function, helps to eliminate postoperative gas pains, and prevents a patient from becoming unnecessarily weak. It is important in getting a patient up on the day of operation or on the following day to apply an abdominal binder for support and to raise the back-rest of the bed to a full sitting position before assisting the patient to stand on the floor. In this way there is minimum strain on the muscles and minimum discomfort.

Repair of bilateral inguinal hernias may be carried out at one operation provided there are no complications. It is our policy to do both sides at one time in the case of indirect hernias, but usually to wait five to seven days between operations for direct or femoral hernias.

Anesthesia is ordinarily not a difficult problem. Intravenous pentothal—curare supplemented with nitrous oxide—oxygen gives very satisfactory results. In poor risk patients, local infiltration of novocain may be used and in certain cases spinal is preferred. With any type of anesthesia it is, of course, essential to have adequate relaxation and minimum tension during the course of the muscle repair.

Results of the treatment of groin hernias in this way have been gratifying. Recurrences will always occur and more particularly in large direct and femoral hernias where the muscular support is poor. However, we have not seen any recurrences among

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our patients operated on by the Cooper's ligament technique during the past three years. These patients all have had femoral or direct hernias, some in debilitated individuals with large muscle defects. With a relatively short period of follow-up, it would be foolish to claim a very high cure-rate, and yet the outlook for successful results appears good at this time.

Complicated hernias are seen occasionally, and these require special techniques. The most baffling type seems to be the sliding indirect hernia. Cure of this type necessitates complete removal of the sac without damage to that portion of bowel which forms the posterior wall. Employment of the abdominal approach and reconstruction of the defect in the mesocolon enable one to accomplish this, and then repair of the canal proceeds as in the ordinary types.

Incarcerated hernias are sometimes reducible by gentle taxis. Reduction *en masse* is to be feared in this type of hernia, and as a rule emergency operation is preferred whenever the hernia cannot be reduced after a few minutes of gentle effort by the physician. General anesthetics may ordinarily be employed in incarcerated hernias, but in long-standing cases it may be necessary because of the patient's poor condition to use local anesthesia. If strangulated viscera are found in an incarcerated hernia, they must be resected or treated immediately and the hernia repaired concomitantly. It is wise to do the simplest form of repair in the case of incarcerated or strangulated hernias, particularly in the older age group.

Summary

Surgical repair of groin hernias is an exercise in technique which is excellent for training and for review of fundamental principles.

Indirect hernias require complete removal of sac and "standard" type of repair.

Direct hernias should be repaired by Cooper's ligament technique. This is also used for femoral hernias.

Employment of the proper operation for the various types of hernia is essential for successful results.

805 West Franklin Street.

Describes New Type of Surgical Needle.

A new type of surgical needle, for use in suturing incisions of the mitral valve of the heart, was described in the January 10 J.A.M.A. by Dr. Gerald H. Pratt, New York.

A ski-shaped type of needle, the new addition to the physician's armamentarium, was made with the base of the ski as a long square, instead of a flat surface, as is the case in most surgical needles. This change, Dr. Pratt stated, permits the needle to be held securely at any angle and makes suturing of the heart valve less hazardous.

"The use of such a needle for other deep suturing is also feasible," he added. "Other parts of the heart, the deep lying vessels in the chest or abdomen, the bronchi, the pelvic bowel and the bladder at times all present suture problems in which such a needle proves valuable."

Dr. Pratt is associated with the vascular clinic of St. Vincent's Hospital and the New York University College of Medicine.

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BENIGN GYNECOLOGIC BLEEDING

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Abnormal vaginal bleeding is probably one of the most frequent conditions which the general practitioner as well as the gynecologist meets in his practice. It is certainly one of the commonest reasons for pelvic examination. The causes may be functional, or they may be various neoplastic diseases. It may also be associated with inflammatory conditions, certain constitutional diseases, retained gestational products, intra-abdominal hemorrhage or uterine bleeding due to various causes.

During the past five years (1947-1951) there were 5955 cases of gynecologic discharge at the New York Polyclinic Hospital. Of these hemorrhage was a predominant symptom in 2354 cases, among which were 752 cases of fibroids, 79 cases of adenomyosis, and 57 cases of tubal pregnancy. The accompanying table indicates in further detail all cases in which hemorrhage was an important symptom. Hemorrhage also occurred as a secondary post-operative complication in 9 cases, including both laparotomies and vaginal plastic repairs, in which proper ligation and hemostasis were not obtained. The commonest pathological finding associated with abnormal vaginal bleeding was leiomyoma of the uterus. Cervical and fundal polyps were the next most common, followed by endometrial hyperplasia and adenomyosis. Pelvic inflammatory disease was established by pathological examination in 16 cases, and ruptured corpus luteum in 7 cases.

It is essential that every patient presenting a history of abnormal vaginal bleeding should be correctly diagnosed, especially with regard to differentiation between benign and malignant disease. For this reason each case should be thoroughly examined. The plan of investigation should be well organized, and fully adequate in all studies requisite to diagnosis of the individual case. A very accurate history of the bleeding should be taken, particularly emphasizing the duration of bleeding and the amount of blood lost. The patient's estimate of blood loss especially will often not fit in with clinical findings or laboratory data, but when it does agree this evidence is a valuable aid in diagnosis.

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FUNCTIONAL BLEEDING

Abnormal uterine bleeding may be regarded as functional when no general or local lesions can be found. It may occur during adolescence, during the reproductive period, at the menopause, or later, but age should be considered in the diagnosis since other factors are more likely to occur in some age groups than in others. Excessive flowing in the adolescent girl and young married woman, for instance, is usually due to a functional disturbance. In a married woman during her reproductive period excessive flowing may be of functional origin, but it is more commonly due to such other causes as complications of pregnancy, infections and benign tumors. In the premenopausal woman functional excesses of flowing are common, but malignancy of the cervix and body of the uterus must be ruled out. Post-menopausal bleeding is due to malignancy in

TABLE I
CAUSES OF BLEEDING

Fibromyomata uteri	752
Incomplete abortion	681
Cervical polyps	219
Hyperplasia of endometrium	198
Threatened abortion	99
Adenomyosis	79
Endocervicitis	65
Ectopic Pregnancy	57
Endocrinopathies, including functional uterine bleeding	43
Endometrial polyps	43
Ovarian cysts	33
Endometriosis	17
Infections	16
Fibrosis of uterus	13
Spontaneous complete abortion	10
Corpus luteum cyst	7
Hydatidiform mole	6
Bleeding following panhysterectomy	5
Missed abortion	3
Bleeding following pelvic floor repair	2
Bleeding following anterior colporrhaphy	1
Bleeding following amputation of cervix	1
Laceration of vagina	1
Papilloma of vagina	1
Granuloma of vagina (non-specific)	1
Hypertension	1
Total	2354

about 50% of cases, but it may also be due to overtreatment of menopausal symptoms with estrogens. A diagnostic curettage is not necessary for the adolescent girl or the young unmarried woman, but in a woman 35 years of age or older it should be done to rule out possible malignant disease. Functional bleeding in women of the child-bearing age should always have a diagnostic endometrial biopsy.

According to Allen[1] the term functional bleeding can be applied to virtually all disturbances of ovarian function in which abnormal uterine bleeding occurs regardless of the microscopic appearance of the endometrium. The histologic appearance of the endometrium is believed to indicate the type of hormonal influence to which it has been subjected only so long as the ovaries are functioning normally. That is, a progestational endometrium indicates that the ovaries are producing progesterone, and a well-developed mid-interval endometrium indicates that they are producing estrogen. However, when abnormal function occurs the normal sequence of estrogen and progesterone production in normal amounts is disturbed and the endometrium becomes bizarre.

Estrogens have been shown to act in relation to bleeding in two ways. That is, too much of the hormone used over too long a period of time may cause bleeding, or abrupt withdrawal may also cause bleeding. This has been established by investigations which show that estrogens when given to ovarectomized or menopausal women for a period of three or four weeks produces a growth of the endometrium. But if the same dose is continued for another three or four weeks bleeding is liable to occur despite continuation of the estrogen. Beginning about five to eight days after estrogen has been discontinued, bleeding may also occur, due to hormonal deficiency. Hence the term withdrawal bleeding.

The most commonly used hormones in the treatment of functional vaginal bleeding are estrogen and progesterone. The latter is usually used according to a withdrawal plan. It has, however, been found that testosterone does not control bleeding any more rapidly than estrogen. Moreover, testosterone is not only more expensive, but the large doses required to stop bleeding may lead to prolonged occurrence of such unpleasant symptoms as acne, hirsutism, gain in weight, hypertrophy of the clitoris, and voice changes. Because of these dangers and because the same beneficial results can be obtained by the use of larger doses of estrogen, the author prefers estrogen to progesterone therapy. In the relatively small number of ovulatory rather than anovulatory functional bleeding cases the causes are unknown and apparently vary both in the individual at different times, and between different individuals.

Functional bleeding in adolescent girls is nearly always due to anovulatory behavior of the ovaries. The decision of when to treat and when not to treat an adolescent girl for abnormal bleeding is not too difficult, for we all know that infrequent periods require no treatment beyond reassurance. Even those patients who have an anovulatory period every two weeks need no therapy unless the bleeding is profuse and several short cycles have occurred, since many patients will have a few short cycles and then will spontaneously revert either to infrequent cycles or regular cycles. These irregularities are usually associated with disturbances in pituitary and ovarian function, and characterized by failure of ovulation—resulting in sterility and bleeding from interval or estrogen prepared endometrium. We have been following the plan suggested by Hamblen[2] in the treatment of these cases. Daily doses of premarin ranging from 3.75 to 7.5 mg. are prescribed. The underlying disturbance may be corrected by cyclic estrogen-progestin therapy continued after hemostasis has been effected. Whatever dosage of premarin was required for hemostasis, the same dosage should be continued for twenty days after hemostasis. During the last ten days of this treatment, pranone is also given by mouth. If the daily dose of premarin was 7.5 mg., the daily dose of pranone will be 60 mg. As a rule when the first cycle of estrogen-progestin therapy is concluded there will be an onset of bleeding within a few days. On the third to fifth day of this episode of bleeding another cycle of estrogen-progestin therapy is begun, and is given similarly save for the fact that a lower dosage schedule is used, that is, a daily dose of 3.75 mg. of premarin and a daily dosage of 30 mg. of pranone. In all, two or three similar cycles of treatment are advisable. This form of therapy regulates bleeding quite promptly, and is followed in the majority of patients by a resumption of normal ovarian and pituitary functions which are evidenced by normal ovulatory cycles com-

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patible with fertility. Such therapy saves many young women from repeated curettages, radium, Roentgen therapy or even hysterectomy.

In cases of ovarian deficiency in young women, the pituitary-gonadal relationship is apparently upset by emotional stress, general poor health, hypothyroidism and perhaps other factors. General supportive measures such as the proper vitamins, iron and thyroid should be used, and are an important adjunct to hormonal treatment during the acute phase. Metabolic studies should also be made, since it will obviously be impossible to cure ovarian dysfunction in a patient in whom the cause is thyroid deficiency unless this deficiency is treated.

In women 35 years of age or older, when endocrine therapy fails, a satisfactory treatment is the intrauterine application of radium. A dose of 1800 to 2500 milligram hours will usually stop the bleeding. We recommend this treatment rather than irradiation with Roentgen rays. If endocrine therapy is not satisfactory in the treatment of women of the child-bearing age, however, a curettage and intrauterine application of radium from 300 to 500 milligram hours is indicated. A higher dosage is contraindicated since it has been found that 600 milligram hours comes within the danger zone of sterilization.

NEOPLASTIC DISEASES

As will be seen from the accompanying table, the commonest of the benign neoplasms causing bleeding were the myomas. However, it is essential to realize that myomas do not themselves cause bleeding unless they are submucous, and do not bleed after the menopause. We must therefore look for polyps or other causes for the bleeding in myomas; failure to do so has caused many deaths. As Karnaky[3] has pointed out, excessive bleeding in a myomatous uterus may be effectively treated as dysfunctional bleeding in a fibroid uterus. It must be emphasized that unless the myoma encroaches upon or invades the endometrium it will not cause bleeding.

For women in the late thirties or early forties irradiation should not be employed since a sterilizing dose is necessary if the myoma is to be cured. An inadequate, non-sterilizing dose will temporarily relieve the bleeding but the myoma usually continues developing. For women in the late fifties, however, radium is very successful in controlling bleeding due to small intermural growths. A sterilizing dose must, of course, be given in order to destroy the ovaries. We have found radium preferable to

TABLE II

CAUSES OF BLEEDING DURING PREGNANCY

Abortions		793 cases
Threatened	99 cases	
Spontaneous (complete)	10 "	
Missed	3 "	
Incomplete	681 "	
Ectopic Pregnancy		57 "
Total		850 "

TABLE III

CAUSES OF BLEEDING AND MANAGEMENT OF CASES IN 1149 CASES WITH BENIGN PELVIC NEOPLASMS

Cause	Number of Cases
Fibromyomata uteri	752
Hysterectomy	734
Myomectomy	17
Radium	1
Cervical and endometrial cysts	262
Dilatation and curettage	260
Biopsy and polypectomy	2
Ovarian neoplasms and cysts	33
Laparotomy	33
Conservative surgery	22
Endometriosis and adenomyosis	96
Laparotomy	96
Hydatidiform mole	6
Dilatation and curettage	5
Hysterectomy	1
Total	1149

Roentgen ray therapy for these cases. Surgery is, however, indicated as a general rule for bleeding myomatous uteri. During the child-bearing years myomectomy should be considered, but if the myoma has too extensively invaded the uterine musculature extensive hysterectomy is necessary, conserving the ovaries in young women.

Cervical and fundal polyps consist usually of a polypoid type of endometrial hyperplasia. These bleed easily, and "spotting"—the term ordinarily used by the patient—is usually noted after coitus, douches, exercise, or it may appear as menorrhagia at the time of her period. The author has not found radium entirely satisfactory in treating polyps, but finds them best handled by curettage or surgery.

Adenomyosis is due to the invasion of the musculature of the uterus by endometrial glands. The uterus thus hypertrophies, enlarging symmetrically. Ab-

normal bleeding is the most important symptom, and usually appears as profuse menorrhagia without intermenstrual flow. Failure of radiation therapy to cure the bleeding is diagnostic. Surgery is indicated.

In general, benign ovarian neoplasms are not among the common causes of bleeding, but the following should be looked for. Cystic degeneration of the ovaries may lead to either prolonged menstruation or to spotting due to imperfect formation of the corpus luteum, especially if there is a corpus luteum cyst. Simple follicular cysts, however, do not as a rule cause menstrual disturbance. Granulosa cell tumor, on the other hand, is frequently associated with bleeding, and should be suspected in cases of post-menopausal bleeding with slightly enlarged uterus and hyperplasia of the endometrium revealed by biopsy. Only about 12 per cent of patients with dermoids have menstrual disorders, which also holds true of those with pseudomucinous cystadenomas. Ninety-six patients with endometriosis or adenomyosis had excessive or abnormal bleeding. In those cases that complained of pain with bleeding, laparotomy was done.

One of the commonest causes of excessive and prolonged bleeding is the retention of products of gestation following labor or abortion. This should be kept in mind in cases revealing delay in the previous menstrual period. Our group included 684 such cases. The private cases were treated conservatively, but on the general service uterine curettage was performed in 75%. In ectopic gestation, spotting and metorrhagia is almost always associated with other characteristic symptoms so that diagnosis presents no serious difficulties.

INFECTIONS

Bleeding due to infections such as salpingoophoritis or cervicitis usually subsides with the control of the infection. Antibiotics and chemotherapeutic agents have eliminated many of the complications which attended these conditions in former days, but cases of cervicitis should nonetheless be biopsied because early carcinoma is not clinically distinguishable from benign cervicitis. If no malignancy is found, treatment is best carried out by means of actual cautery and douches.

INTRA-ABDOMINAL HEMORRHAGE

Ectopic pregnancy is the commonest cause of intra-abdominal hemorrhage of genital origin. Bleeding may vary from slight to profuse. Intra-abdominal hemorrhage may also be due to rupture of the pedicle of a pedunculated myoma, or from rupture of a corpus luteum cyst, as reported by Sackett[4] and Morton[5]. Hemoperitoneum from this cause is not uncommon, though it is most commonly misdiagnosed as acute appendicitis, ruptured extra-uterine pregnancy or salpingitis. Morton, in 1931, for instance, collected 26 cases of ruptured corpus luteum and found that in all of them the condition had been misdiagnosed. Follicle rupture, or Mittelschmertz, is usually recognized, while corpus luteum rupture is seldom considered even as a possibility. This larger error in diagnosis is probably due to the lack of appreciation of the clinical entity of the condition.

Rupture of the corpus luteum may occur at any time during the child-bearing period from the onset of puberty to the menopause, but is commonest in the young adult. While rupture of the Graafian follicle occurs characteristically during the mid-interval period, that of the corpus luteum occurs during the second half of the interval from the 18th to the 24th day of the cycle. Pain is a constant symptom and varies in intensity depending on the amount of hemorrhage, but is usually sudden in onset and confined to the lower part of the abdomen. Nausea may be present, but rarely vomiting. Such sudden onset of abdominal pain during the second half of the menstrual cycle in a woman whose periods have been regular is suggestive of ruptured corpus luteum. Soft, flat, non-rigid but tender abdomen with a definite rebound tenderness strongly suggests free blood in the abdominal cavity. Bleeding is seldom extensive enough to cause shock.

An acutely inflamed appendix, co-existing with a ruptured corpus luteum has been reported by O'Gorman[6] and by Hoyt and Meigs[7]. Such coincidence can, of course, happen. Surgery is employed for ruptured corpus luteum cysts, conserving the ovary if possible. In case of ovarian cysts the cyst lining is removed and the ovary sutured.

MISCELLANEOUS CAUSES

Among the miscellaneous causes of benign gynecologic bleeding may be mentioned senile vaginitis. Hypertension producing arteriosclerosis of the uterine vessels may also cause bleeding. Diseases of the thyroid may also be responsible for the condition.

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Constitutional diseases are, however, rarly causative of vaginal bleeding, although pneumonia, influenza, chronic valvular disease, diabetes, hemophilia, scurvy and syphilis have some tendency to produce menorrhagia. In all these conditions the treatment is to remove the cause rather than to treat the secondary symptom.

TRAUMA

Injury of the external genitalia is a frequent cause of hemorrhage, especially if the venous plexus of the labia are lacerated. Occasionally a troublesome hemorrhage may follow a plastic operation on the cervix, vagina or pelvic floor. The author has seen three cases of such severe hemorrhage that suturing and transfusion were required on the tenth day after amputation of the cervix.

SUMMARY

1. An analysis has been made of patients with benign gynecologic bleeding in the New York Polyclinic Hospital during the years 1947-1951.
2. Early complete and correct diagnosis is important, and every patient with abnormal vaginal bleeding should be examined thoroughly.
3. Biopsy should be done on any questionable cervical or vulvar lesion.
4. Endocrine therapy may be used in treating patients with benign vaginal bleeding. Estrogen-progesterone treatment has been found effective.
5. Endometrial curettage is a diagnostic necessity in many cases, and should be used therapeutically before attempting further treatment of irregular bleeding.
6. Physicians as well as women should be alerted to the danger of irregular bleeding at the menopause. The possibility of malignancy should be kept in mind until ruled out.

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116 East 68th Street.

Fourth Supplement to Motion Picture Reviews Now Available.

The Committee on Medical Motion Pictures of the A.M.A. has completed the fourth supplement to the booklet entitled "Reviews of Medical Motion Pictures." It contains all the film reviews published in The Journal, from January to December 1952.

The purpose of the reviews is to provide a brief description and evaluation of motion pictures which are available to the medical profession. Each film is reviewed by competent authorities.

One copy has been mailed to the secretary of each state medical society. Copies are available to county medical societies on request from: Committee on Medical Motion Pictures, American Medical Association, 535 North Dearborn Street, Chicago 10.

CERTAIN REPARATIVE SURGICAL PROCEDURES OF THE UPPER RESPIRATORY TRACT*

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The otolaryngologist is being constantly consulted by patients whose complaints, among many others, are related to the respiratory passages. Many of these complaints are due to obstruction to the upper air passages by acute inflammatory and allergic conditions, neoplasms, functional disorders, or to structural changes due to congenital abnormalities, accidental trauma, surgical trauma, and cicatricial changes secondary to disease or corrosives. It is in the group of structural changes that we are interested at this time. We wish to bring briefly to your attention some common but more often uncommon disorders obstructing the nasal, pharyngeal, and laryngeal air passages, that we have encountered.

By far the larger number of cases are those of trauma to the nose, resulting in external and intranasal deformities. A badly injured nose which has had unsuccessful primary care can be unsightly and result in much discomfort to the owner when the airway is obstructed because of the disorganized bony, cartilaginous, and soft tissue components. Figure 1a shows an example of such a case with external nasal deformity, scarring of the soft tissues of the right vestibule, and almost total obstruction to the left side intranasally because of displaced septal cartilage and bone. This was not a neglected case; as good a primary result as possible, under the existing conditions, was obtained. To correct the deformity will require skin grafting and the removal and replacement of various cartilages and bones of the nose, and is no simple problem. The correction of the deformity as illustrated in Fig. 1b is less difficult.

A patient who has experienced adequate intranasal air space will suffer considerable discomfort when deprived of this rather suddenly by trauma. Satisfactory correction can be obtained only by the more definitive rhinoplastic surgical procedures. The common "classical submucous resection" has not sufficed and will not suffice in these particular cases. In some incidences, septal reconstruction without alteration in the appearance of the nose is possible, but in many cases is not practical nor justifiable. With the increase in nasal injuries and in laity consciousness of so-called "plastic surgery," the necessity for various rhinoplastic procedures, rather than simple septal resections, becomes apparent. Though irrelevant to this paper possibly, we cannot help but make the observation that nasal bone fractures are

Fig. 1 a and b.—Two nasal deformities resulting from trauma which will require corrective rhinoplastic surgical procedures.

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*Read before the annual meeting of the Medical Society of Virginia, at Richmond, September 28-October 1, 1952.

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probably given less consideration by the medical practitioner than any other fracture.

Congenital atresia of the posterior choanae is not common. The obstruction may be unilateral or bilateral, membranous or bony. Figure 2 shows a diagram of a unilateral atresia, as visualized from the nasopharynx. Bilateral atresia in the infant is

Fig. 2.—Diagram of right posterior choanal atresia.

an emergency problem. Failure in recognition and relief may result in asphyxia neonatorum, and, if not this, then malnutrition because of inability to feed properly. Unilateral obstruction in the older child or adult is disturbing and embarrassing because of the constant presence of secretion in the vestibule and anterior nares, of which the patient can rid himself only by postural changes or by some mechanical means. Peculiarly enough, the inability to breathe through one side of the nose does not cause as much discomfort as one would imagine. To establish an initial opening in the obstruction is no problem, but to maintain its patency is a most difficult one. Here, as in many cases, the vexing problem of circumventing the scar tissue changes is encountered.

Several different types of operations, varying as to the surgical approach, have been proposed and advocated by various rhinological surgeons. The intranasal approach, with perforation of the obstruction and insertion of a cannulated obturator of some type, is used most often as a temporary measure in infants, to be followed later, as necessary, by a procedure designed to give improved results. In these later operations, the intranasal, transantral, or transpalatine approach is utilized to remove the obstruction plus bone of the septum and the palate. Care is taken in the handling and replacement of the soft tissues to minimize future scarring. The transpalatine approach offers the advantage of attacking the problem with more available space and under direct vision. Figure 3 illustrates a step in the transpalatine approach.

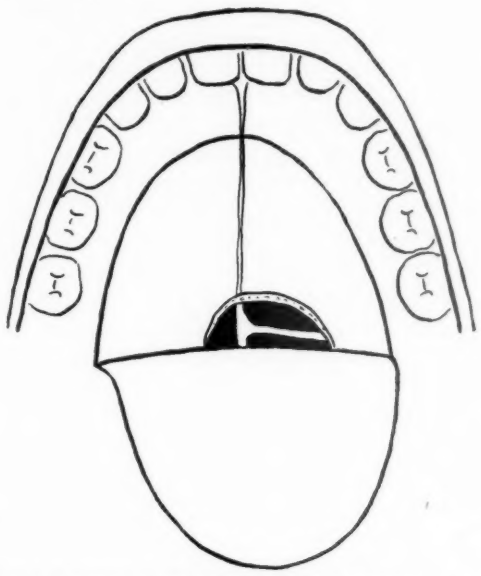

Fig. 3.—Diagram illustrating transpalatine approach in correction of choanal atresia.

Scarring and adhesions of the tissues of the soft palate, pharyngeal wall, and tongue, resulting to a degree in incomplete stenosis and respiratory hindrance, are rare. At the present time the cause is usually secondary to surgery, such as tonsillectomy and adenoidectomy. Caustic burns, syphilis, rhinoscleroma, and other conditions have been reported as other causes. The symptoms will obviously depend upon the degree of the stenosis and location. Successful correction of the condition apparently is most difficult. The scar tissue must be removed and the resulting lumen retained by means of dilatation, obturators, and skin or mucous membrane grafts. We have had experience in the treatment of only one case, a child of five years of age with an incomplete glossopharyngeal stenosis, secondary to tonsillectomy and adenoidectomy. Fortunately, the scarring was not dense and was corrected by, first, radial incisions, and, then, frequent dilatations under general anesthesia. A tracheostomy was performed prior to treatment. Figure 4 illustrates diagrammatically the sites of the obstructions.

Stenosis of the airway above the laryngeal level is rare, compared to that at the level of the larynx.

We have encountered this latter group of cases surprisingly often, even with the reduction in the incidence of diphtheria, syphilis, and tuberculosis. Violent trauma to the cervical region with subsequent scarring has been the source of our experience in several recent cases, and tuberculosis in another. As yet we have had no cases secondary to endotracheal or endoesophageal intubation procedures, which have become very popular in recent years and will un-

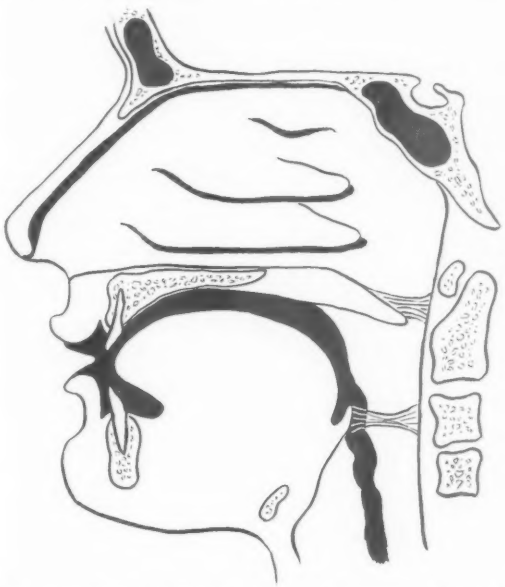

Fig. 4.—Diagram illustrating sites of pharyngopalatine and pharyngoglossal atresias.

doubtedly yield material of the type in which we are interested. Ten years ago, at one period, we had under our care, for rehabilitation, four children with laryngeal stenosis secondary to diphtheria, intubation, and, in one incidence, misplaced tracheostomy. All were decannulated and now have good airways and voices. Treatment consisted of repeated progressive dilatation of the intralaryngeal lumen. No small factor in the successful results is the increase in the relative size of the laryngeal structures with the growth and development of the child.

More recently our efforts have been directed to reconstruction of the badly disorganized laryngeal tissues secondary to trauma. In addition to airway, speech (voice) must be considered in the evaluation of the ultimate result. Again, the problem of ever recurring scar tissue is encountered. In repairing these conditions, indwelling tubes or molds of rubber or plastics, with and without grafts, are utilized. The grafts may be of skin or mucous membrane. When more space is necessary, bone and cartilage (hyoid bone and septal cartilage) have been used as a midline graft. Figure 5 illustrates this point.

Another cause of embarrassment in the size of the glottic space is bilateral abductor paralysis of the vocal cords. This usually is secondary to thyroid surgery for goiter or carcinoma. As the result of the

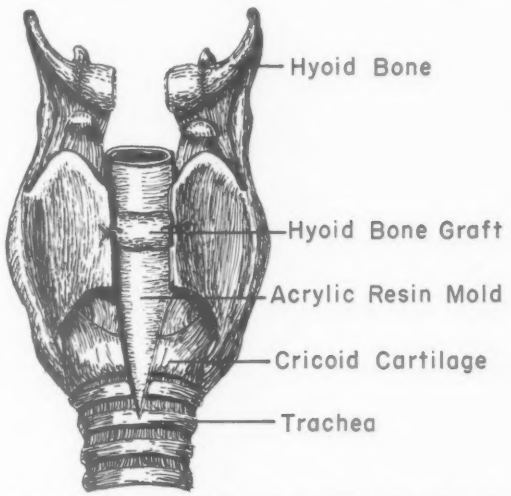

Fig. 5.—Diagram illustrating use of hyoid bone graft and acrytic mold in reconstructions of laryngeal airway.

injury to the recurrent laryngeal nerves, the paralysed vocal cords are immobilized in a position very near the midline of the larynx, obstructing the airway, but not interfering with the voice. A tracheostomy is generally necessary within a few hours following the thyroid surgery. Incidentally, the tracheostomy tube should not be removed until a competent otolaryngologist has given an opinion as to the motility of the vocal cords. If immobility persists in each cord, then obviously the patient is safer with the additional airway afforded by the tracheostomy.

For the correction of this condition, we have a precise and successful operative procedure. By submucosal dissection, one arytenoid cartilage is removed, usually by an external approach. Thus, the glottic space is increased with little, if any, sacrifice of voice. The patient may be safely decannulated, or, in the event that a tracheostomy has not been deemed necessary, can live a safer and more effortless existence. Note Fig. 6.

Finally, there are two other conditions met with

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infrequently that cause respiratory effort in children. Our experience with these has been limited to one case of each type. Micrognathia or small lower jaw with inadequate tongue support will sometimes seriously interfere with respiration and feeding in infants. An operation (Douglas) has been devised to relieve the obstruction by bringing the tongue forward and fixing it to the alveolar ridge and lower lip, thus improving the airway and also encouraging the development of the jaw. The second condition is the rare congenital webbing of the larynx. Our case required only dividing a thin web between the anterior one-third of the vocal cords. Other cases may be very difficult problems.

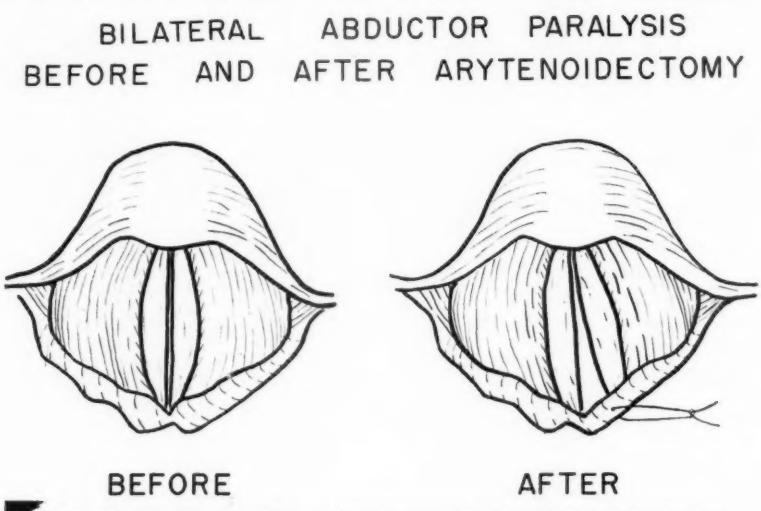

Fig. 6.—Diagram illustrating bilateral abductor paralysis before and after arytenoidectomy.

SUMMARY

Several conditions, usually structural in nature, of the larynx and upper respiratory tract, resulting in respiratory difficulty, are mentioned. It has been indicated that reparative surgical procedures are effectual in the correction of these conditions. In many incidences a beneficial solution of the problem is difficult, but an improved result may be expected in the majority of cases.

104 East Market Street.

A Manual for Arthritis Clinics,

Said to be the first of its kind, is just off the press from the Arthritis and Rheumatism Foundation, according to an announcement by the national medical director, Dr. Gideon K. deForest. The manual was published jointly with the National Institute of Arthritis and Metabolic Diseases, and is a 64-page book.

Priced at 50 cents, copies may be obtained by writing to any of the 32 Chapters of the Foundation or to national headquarters, 23 West 45th Street, New York 36, N. Y.

ASPIRIN POISONING*

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Richmond, Virginia

This paper is written not so much with the expectation of presenting new material but with the hope of bringing to the attention of the practicing physician the seriousness of aspirin poisoning in children. Consideration is given to the toxicity due to over-dosage, taken either accidentally or therapeutically, and not to aspirin hypersensitivity. In a review of the more recent American literature[2-11] 77 cases of salicylate poisoning were found. Of these 46 were given the drug therapeutically and 31 took it accidentally. There were 11 deaths.

The following case came under my observation on March 3, 1951.

CASE REPORT

L. C., white male infant, 18 mo. of age, weighing 25 lbs., was seen at 3:30 A.M. at home. He was breathing rapidly in a deep, pauseless manner. He looked pale and lethargic but could be roused and appeared conscious. The history obtained from the practical nurse was that 18 hours earlier, between 8-9 A.M. of the previous morning, he had eaten 15-20 aspirin tablets, each 1¼ gr. (20-25 gr.). He had seemed all right after this, had had a normal day, had taken his usual meals and had gone to bed in good condition. During the early morning when the nurse was taking care of another child, she noticed the child breathing strangely and had called the physician. The child was taken immediately to the hospital.

On admission the temperature was 103° F, pulse 150, respiration 60. The skin was warm and dry and, except for thick saliva in the pharynx, the physical examination was not revealing. Urine obtained was acid and showed acetone. The child was put immediately into an oxygen tent and 360 cc. of 1/6 molar sodium lactate solution with the addition of alidase was given by clysis. This was promptly absorbed. Orange juice with 100 mg. ascorbic acid added was given by mouth. Ten milligrams of vitamin K was given parenterally. Sponging for fever and elixir phenobarital drachms 2 for twitching were administered. At 10:30 A.M. the patient began to spit up coffee ground fluid and was comatose. A vein was cut down upon and an additional 200 cc. 1/6 molar sodium lactate were given I.V., followed by a transfusion. Despite all measures tried, including stimulants, the patient went continuously down hill and died at 12:55 P.M.—approximately 9 hours after admission. His temperature toward the end reached 105.8°F. His respirations continued in the deep, rapid, pauseless manner until near the end when they became more gasping.

Laboratory Reports:

B1. count: Hgb. 14 gm., R.B.C. 4,440,000, W.B.C. 26,400, P.M.N. 34, Lymph. 58, Mon. 8. B1. Chlorides: 580 mg./100. CO_2 combining power 30.5 vol. per cent (done on ½ cc. hemolyzed blood.) Blood salicylate level: 75 mg./100.pH 7.30 (venous blood) done 1 hr. before death. Prothrombin time 19 sec. (control 13 sec.).

AUTOPSY

(Head not done) Findings of significance were: Grossly; Petechial hemorrhages on the epicardial surface of the heart with moderate dilation of both ventricles; areas of hemorrhagic consolidation in both lungs; petechial hemorrhages in the submucosa of the stomach. Liver yellow in color and mottled in appearance.

Microscopicly: Diffuse congestion of sinusoids of liver; diffuse congestion of spleen with nuclear debris in germinal centers; focal erosion of stomach mucosa with superficial necrosis; lymph follicles of colon and appendix prominent with germinal centers showing nuclear debris and necrosis; some hemorrhage in periadrenal fat and focal lipoid depletion; some cloudy swelling of convoluted tubules of kidneys with congestion; prominent follicles with necrosis of lymph nodes; necrosis and calcification of periarterial connective tissue in heart; hemorrhagic areas in the lung.

In summarizing the literature at our disposal certain facts are evident.

Symptoms:

The time of appearance of the symptoms of in-

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*Read before the annual meeting of the Medical Society of Virginia, in Richmond, September 28-October 1, 1952.

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toxication depends upon the method of ingestion—whether in supposedly therapeutic dosage or in accidental large amounts. After accidental ingestion of toxic amounts symptoms do not appear for 12-24 hours. When given in divided doses there is an accumulative effect and symptoms appear when toxic blood levels are reached, varying from 1 to 4 days. The symptom which is constantly found is hyperpnea. This at first is mild but becomes increasingly severe until the deep, pauseless breathing of the Kussmaul type is reached. This continues until the causative acidosis is corrected or until the patient dies of respiratory failure. Other symptoms frequently found are: vomiting which may contain coffee ground material, an acetone odor to the breath, irritability and restlessness, cyanosis or pallor, semiconsciousness or stupor, profuse sweating, dehydration, often fever of high degree, not infrequently convulsions and abdominal pain. Occasionally there is actual bleeding from the mucous membranes and a purpuric rash. Scanty urine, progressing to anuria, occurs as the intoxication increases. In older patients the subjective symptoms of tinnitus and dizziness and paresthesias may be complained of and signs of mental confusion and delirium may occur.

Laboratory Findings:

The laboratory findings vary with the degree of intoxication and the time of study. A constant finding is salicylate in the blood and the urine. The amount of salicylate in the blood which is required to produce symptoms of poisoning must vary with individuals. As small an amount as 29 mg. % has been reported producing intoxication, while one patient with a blood level of 68.4 mg. % recovered after heroic treatment. One patient who died had a level of 86 mg. % and the present reported case had 75 mg. %. There may be a reduction of blood sugar as low as 44.8 mg. The CO^2 combining power is always reduced in the acidotic stage. Cases as low as 14 vol. % are reported. A blood pH 7.1 has been recorded. The prothrombin time is frequently increased. The highest one was 2 min. 45 seconds. The longest bleeding time was 10 min. with clotting time 7½ min. A platelet count of 42,000 has been reported. The urine may be alkaline or acid depending upon the period of observation after intoxication begins. When acidosis has begun acetone and frequently diacetic acid are found.

Pathological Physiology:

To understand these symptoms and laboratory findings, a knowledge of the underlying disturbed physiology is necessary. In 1937, Dodd *et al.*[1] reported on the effects of salicylates on animals. They showed that the direct effect is an increase in metabolic rate with increase in the dissipation of heat, nausea and vomiting, irritation of the kidneys, and more or less mental disturbance. The vomiting, diuresis and sweating produce dehydration with a rise of temperature and decrease in urinary output. An acidosis develops secondary to the inadequacy of the circulation and the production and accumulation of ketone bodies, which are the result of the loss of food reserves of the body incident to the rapid metabolic rate. Dodd states that "no serious symptoms are produced by the administration of large amounts of salicylates to experimental subjects as long as the intake of fluid is adequate and there is no interference with the processes of the elimination of heat". It was Hartman and his associates[2] in 1942 and 1945 who most clearly outlined the process of salicylate action in excessive amounts in human subjects. The course of events can be divided into three stages, each having its definite characteristics and each requiring entirely different procedures to counteract.

The first stage is one of alkalosis. The salicyl radical acts directly on the respiratory center, producing an hyperpnea. There is a respiratory loss of CO_2, altering the ratio of carbonic acid to bicarbonate in the direction of alkalinity, thus producing a respiratory alkalosis. To compensate bicarbonate is excreted in the urine with a resulting alkaline urine.

The second stage is one of compensated acidosis. The salicylates act specifically on carbohydrate metabolism with resulting rapid depletion in the glycogen reserves of the body and the production of a metabolic acidosis with ketosis. The alkaline reserve is depleted by the excretion of bicarbonate in the first stage and the compensation is maintained by the continued hyperventilation and the depleted buffers in the blood. The urine is now acid.

The third stage is one of decompensated acidosis. The alkaline reserves of the body are depleted, the buffer system of the kidneys begins to fail, there is a decrease of the pH of the blood—acidemia, and finally circulatory and respiratory failure.

Another evidence of disturbed physiology is the lengthening of the prothrombin time by large doses of salicylates. Stevenson[3], in 1937, in a review of 13 reported autopsies of salicylate poisoning, found that one-half showed petechiae on the epicardium and almost one-third in the pleura and lungs. In experimental studies on six convalescent patients Fashena and Walker[4] showed a marked prolongation of prothrombin time with doses of sodium salicylate, 0.2 and 0.22 gm. per kilo of body weight, which occurred within the first 24 hours and reached its height on the second day. Thereafter there was a spontaneous drop in prothrobin time which, however, did not reach the pre-treatment level. They considered the hemorrhagic complications due to hypoprothrombinemia and other factors, such as vascular dilatation and vascular injury. In a recent paper by Dietrick *et al.*[5], one case was reported with a prothrombin time of 55 seconds and another of 25 seconds (control 17 seconds). Ashworth and McKenie[6] surmise that since the hypoprothrombinemia can be prevented by vitamin K, it is not due to liver damage.

The effect of salicylates in toxic doses on the liver has been demonstrated by Lutwak—Mann[7] to be a depletion of the hepatic glycogen.

Pathology:

Of the 11 deaths, 10 were autopsied. Only one case is duplicated in the review of Stevenson in 1937, that being his own case. Nine of the 10 cases showed the predominating finding of petechial and, occasionally, diffuse hemorrhage. The petechial hemorrhages were wide spread, involving the brain, the lungs, the pleura, the heart and the gastro-intestinal tract, and in some instances the adrenals, the thyroid, the spleen and the lymph nodes. The liver was described as showing parenchymatous degeneration, fatty infiltration, acute inflammatory changes, or hydropic degeneration with beginning necrosis. In some of the autopsies the kidneys were described as hyperemic, or showing fatty degeneration.

Treatment:

From this knowledge of the action of salicylates in the body, treatment depends upon the time the patient comes under observation.

In the case of accidental ingestion of toxic amounts of aspirin immediate gastric lavage with tap water is indicated and hydration forced. It has been shown that salicylates appear in the blood within 10 minutes after ingestion and in the urine in 10 to 20 minutes. The average time to reach maximum blood levels is about 1 hour after ingestion but variations occur up to 12 hours[8]. After hyperpnea occurs gavage is of no value.

During the first stage, the period of alkalosis, the patient should be given intravenous glucose solution in normal saline. The subcutaneous route can be used also. The purpose of this is to spare the glycogen reserves of the body and to combat alkalosis by supplying chlorides to unite with the base present. If the patient is not able to take enough fluids by mouth, intravenous therapy should be continued over a period of time sufficient to promote renal excretion and to combat relapse. At this time, also, vitamin K, 10-25 mg. should be given parenterally to combat the tendency toward hypoprothrombinemia and vitamin C, 100 mg. to help control bleeding. This stage can be diagnosed by CO_2 determinations and the alkaline reaction of the urine.

In the second stage, compensated acidosis, treatment consists of the use of alkalies and glucose solution. The alkaline solution most frequently given is 1/6 molar sodium lactate, although Ringer's lactate is a frequent choice. The amount to be given can be arrived at by the CO_2 determination, and can be given intravenously or subcutaneously, by intravenous drip continuously, or at 6 hour intervals. If CO_2 determinations cannot be made immediately, then the degree of acidosis should be estimated and the lactate solution should be started at once. In addition, 5% glucose in distilled water should be given to supply carbohydrate and to increase hydration and promote diuresis. If the response to the lactate solution has not been satisfactory, some writers reported dramatic response to sodium bicarbonate given in 1.5% solution intravenously in estimated amounts to combat the acidosis. If the patient can take fluids by mouth, orange juice and sodium bicarbonate can be given orally. Vitamin K and vitamin C should be given also subcutaneously. An oxygen tent or O_2 by funnel may be of value and transfusions are helpful, especially if hemorrhagic tendencies are noted. Fluids to cause the elimination of the salicylates in the blood through the kidney are of paramount importance. It has been estimated that 80% of the salicylates appear eventually in the urine as salicyl compounds and 20% as break down products[9].

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Clinically it is hard to be sure of the point at which the third stage, decompensated acidosis begins. From the laboratory standpoint the pH of the blood can make this differentiation. However, from the standpoint of treatment the same conditions exist as in the second stage but more seriously and the treatment should be increased correspondingly. At no point should treatment be relaxed until the patient is well hydrated, conscious and the kidneys are excreting satisfactorily.

If convulsions occur lumbar puncture has been reported valuable and sodium phenobarbital subcutaneously helps control the spasms.

COMMENT

In cases of unexplained hyperpnea salicylate poisoning should be suspected, as it is a constant finding in this type of drug poisoning. In one case reported the history given was that the child was being treated with penicillin and benadryl and not until the second day of hospitalization was it discovered that the parents on their own authority had been giving excessive doses of aspirin to control the fever. A simple qualitative test to determine salicylates in the urine is that of Gerhart's. He showed that when ferric chloride is added to urine a purple ring results and remains on boiling if salicylates are present.

The lethal dose of aspirin for man is not exactly known, but, according to Greenberg[10], is a matter of opinion based on "unavoidably unreliable evidence." Children seem particularly susceptible to aspirin and that is especially so in sick children who are taking little food and fluids. There is a definite accumulative effect, which is increased by the dehydration of fever and vomiting. Because of this it is important to be exact in prescribing aspirin for them. A safe dosage is 1 gr. per year of age every 4 hours or 0.04 gm. per kilo of body weight for a 24 hour period—up to 5 years of age. In rheumatic fever larger doses are given—0.125 to 0.150 gm. per kilo of body weight, to produce high blood levels but these cases should be under strict supervision.

In cases of accidental ingestion of aspirin when the amount is not known it is a wise precaution to have the child lavaged. Today when preparations of aspirin are made attractive to children it is of the utmost importance to warn parents of the danger of over-dosage and to caution them to keep their supply in places out of reach of toddlers. If one has ever seen the distressing appearance of a child in the stage of decompensated acidosis, he or she will not hesitate to employ lavage when an unknown amount of aspirin has been taken.

SUMMARY

1. Aspirin in toxic dosage can cause fatal poisoning.
2. Infants and children seem peculiarly susceptible to relatively small amounts of aspirin.
3. Unexplained hyperpnea in infants and children should suggest salicylate intoxication.
4. Salicylate is always found in the blood and urine to confirm a diagnosis of aspirin poisoning.
5. Hydration and correction of acidosis are the two most important factors in treatment of the acidotic stage.
6. Aspirin has a cumulative effect when given over a period of time.
7. A prescribed dose of aspirin should be exact. A safe dosage for children is 1 gr. per year of age every 4 hours up to five years of age.
8. In accidental ingestion of an unknown amount of aspirin gastric lavage is advocated.

The pathology of this case was reported in more detail in the *Virginia Medical Monthly*, June, 1952, by J. A. Tobin and G. R. Hennigar.

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New Books.

We list below some of the newer books at the Tompkins-McCaw Library of the Medical College of Virginia, Richmond 19, which may be borrowed by our readers under usual library rules.

Adriani—The pharmacology of anesthetic drugs. 3rd ed. 1952.
Advances in carbohydrate chemistry. Vol. 7, 1952.
Albritton—Standard values in blood. 1951.
Baer—Plasma substitutes except those derived from human blood. 1940-51. Annotated bibliography. 1951.
Baldwin—Dynamic aspects of biochemistry. 2nd ed. 1952.
Brock—Lung abscess. 1952.
Ginger, et al., ed.—Bacterial pyrogens. 1952.
Hansel—Clinical allergy. 1933.
Herbut—Urological pathology. Vols. I & II. 1952.
Master, et al.—Normal blood pressure and hypertension. 1952.
McLester & Darby—Nutrition and diet in health and disease. 1952.
Novak—Gynecologic and obstetric pathology. 3rd ed., 1952.
Rennie & Bozeman—Vocational service for psychiatric clinic patients. 1952.
U. S. Army Medical Dept.—The physiologic effect of wounds. 1952.

SAMA Sets '53 Program in Motion.

Strengthening its position as the voice of its 15,000 members, the House of Delegates of the Student American Medical Association set an accelerated program in motion during its second annual convention in December in Chicago.

Chief proposal of the delegates from 50 of SAMA's 59 chapters called for a point system in doctor draft legislation to consider all previous military service, regardless of branch or rank. Another resolution asked for a synchronization of doctor draft calls with hospital training programs so that young doctors will not experience unnecessary delay in resumption of their training following military service. The House also urged its constituent chapters to invite officers and staff members from organized medicine to address local meetings so that students will be kept informed on current medical activities. The next convention is scheduled for June.

XUM

THE HYPERACTIVE CAROTID SINUS*

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In 1799, Parry[1] reported that pressure over the carotid arteries would retard the human heart rate. In 1923, Hering[2] clearly demonstrated that the carotid sinus, located at the bifurcation of the common carotid artery, initiated the reflex mechanism which played such an important role in controlling heart rate and maintaining arterial blood pressure. More recently Weiss and his co-workers[3] defined three types of carotid sinus syndrome, namely: (1) the vagal type producing bradycardia and even asystole; (2) the depressor type in which there is vasodilatation with secondary depression in blood pressure; (3) the cerebral type in which cardiac rate and blood pressure are not appreciably affected, though fainting, dizziness, weakness, drowsiness, numbness and tingling of the extremities, pallor of the face, hypernea, or even convulsions may be the symptoms noted. The first type, which can be blocked by atropinization, is clearly mediated through afferent fibers known to connect the carotid sinus with the vagus and in turn with the cardioinhibitory center in the medulla. The second type is mediated primarily by fibers known to connect the carotid sinus with the superior cervical ganglion of the sympathetic chain. Small doses of adrenalin will abort an attack of this type of over-activity of the carotid sinus, but atropine is without effect. In the third type, the exact neurological site of activity is not known. Adrenalin and atropine are without effect, but procaine infiltration of the sinus prevents attacks previously produced by local stimulation.

Though the exact type of hyperactivity of the carotid sinus may only be of academic interest, this diagnosis can readily be made. If the patient is examined in the sitting position with the neck slightly extended, the bifurcation of the common carotid artery can readily be located just medial and posterior to the angle of the jaw. By massaging the sensitive carotid sinus for ten or fifteen seconds, the syndrome should be reproduced. Should no syncope or other symptoms result from the aforementioned procedure, at least 3 to 5 minutes should elapse before pressure is exerted on the contralateral side. Never should both sinuses be massaged simultaneously. By use of the stethoscope bradycardia or asystole may be readily ascertained. The EKG, though not requisite, may add pictorial and permanent evidence to substantiate the diagnosis. Serial blood pressure readings before, during, and after the period of digital pressure should confirm any hypotension associated with the depressor type. In the cerebral type, syncope without dramatic decrease in heart rate or blood pressure should be reproduced. Though the three characteristic physical signs of the three types of carotid sinus hyperactivity may be present to some degree in any individual case, usually one of these signs will be found to predominate. Two illustrative cases will be presented in the following paragraphs.

*Case 1.** Mrs. P.E.K., a 65 year old widowed, white woman, was hospitalized on 3rd January, 1952, because of upper abdominal pain with nausea and vomiting of three days duration due to cholecystitis with lithiasis. For many years this patient had had an occasional episode of fainting. During the past six to eight months, she had had weakness and syncope associated with flare-ups of her gallbladder disease. Twenty-seven years previously she had been diagnosed as having early toxemia of pregnancy and had been digitalized. She had continued her digitalis since then and was on a maintenance dose of .2 mgm. digitoxin daily. For 15 to 29 years she had recurrent bronchial asthma but had been asymptomatic for about six months.

Physical examination on admission revealed a poorly developed and mildly undernourished woman, who appeared moderately ill and was somewhat dehydrated. The heart was slightly enlarged to the left and downward. The rhythm was regular. The rate was 60. Blood pressure was 120/70. No murmurs were heard. The lung fields were clear. The abdomen was moderately tender in the right upper quadrant with some muscle spasm over the gallbladder area. At the time of her admission, carotid sinus activity was not tested, and the remainder

*Read before the annual meeting of The Medical Society of Virginia, at Richmond, September 28-October 1, 1952.

*Patient reported through the courtesy of Drs. H. L. Archer and E. D. Davis, Jr.

of the physical examination was normal.

Laboratory data: The Kahn test was negative. The hemoglobin was 13 gms., the white cell count was 5,800, and the red cell count was 3.9 million. Admission urine obtained after intravenous glucose administration revealed a specific gravity of 1.018, with a 4 plus reduction for sugar, 4 plus reaction for acetone and a plus 2 albumin. The urinary sediment was normal. A later urine examination failed to show any reduction with Benedict's solution nor was there any acetone or albumin present. The icterus index and prothrombin time were normal.

During the first five days of hospitalization, this lady had exacerbations of her abdominal pain and nausea with vomiting so that she took little by mouth and required repeated infusions. On four occasions, she had episodes of marked fall in blood pressure, weakness, and pallor and a relative bradycardia varying between 60 and 72. These peculiar "shock like" episodes lasted from two to four hours and were relieved by transfusions and/or by the intravenous administration of 5% glucose in normal saline. The fasting blood sugar was normal, and one drawn shortly after an infusion of glucose and during one of her syncopal attacks was 176 mgm. per cent. Examination on the fifth hospital day with carotid sinus massage revealed evidence of a hyperative carotid sinus, primarily of the depressor type with a rather striking drop in blood pressure. The electrocardiogram as well as the laboratory findings showed evidence of hypopotassemia. Figure 1 demonstrates the fall in blood pressure, the decrease in pulse, and the electro-cardiographic changes with carotid sinus massage. Figure 2 presents the electrolytic evidence of hypokalemia and hypochloremic acidosis so often seen in potassium deficiency. It also shows the effect of potassium on the electrocardiographic changes. Her clinical improvement was quite striking. On the seventh hospital day, forty-eight hours after initiating therapy for her potassium deficiency and for her hyperactive carotid sinus, this lady, who had previously had nausea and vomiting, abdominal pain and discomfort, extreme weakness, and syncopal episodes, was feeling much stronger and taking a full liquid diet. Ephedrine sulfate, 24 mgm., four times a day and the equivalent of sixty drops of tincture of belladonna per day were maintained throughout her hospital stay. On the eighteenth day after admission, she had a cholecystectomy without difficulty.

Follow-up visits to the office in April and June, 1952, revealed that she had had no syncopal or weak spells. Her medications had been maintained until about ten days prior to her last check-up. At that

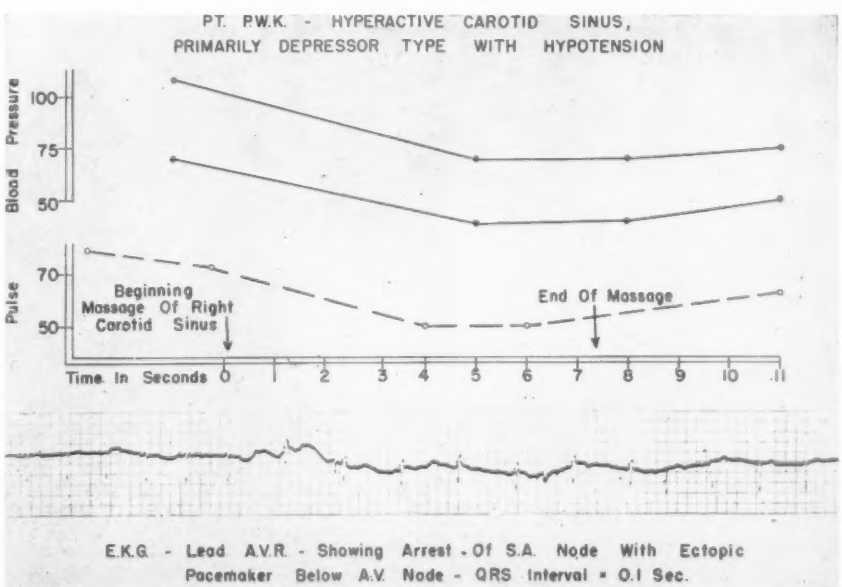
P.T. P.W.K. - HYPERACTIVE CAROTID SINUS,
PRIMARILY DEPRESSOR TYPE WITH HYPOTENSION
Blood Pressure
100
75
50
Pulse
70
50
Beginning Massage Of Right Carotid Sinus
End Of Massage
Time In Seconds 0 1 2 3 4 5 6 7 8 9 10 11
E.K.G. - Lead A.V.R. - Showing Arrest - Of S.A. Node With Ectopic Pacemaker Below A.V. Node - QRS Interval = 0.1 Sec.

Fig. 1

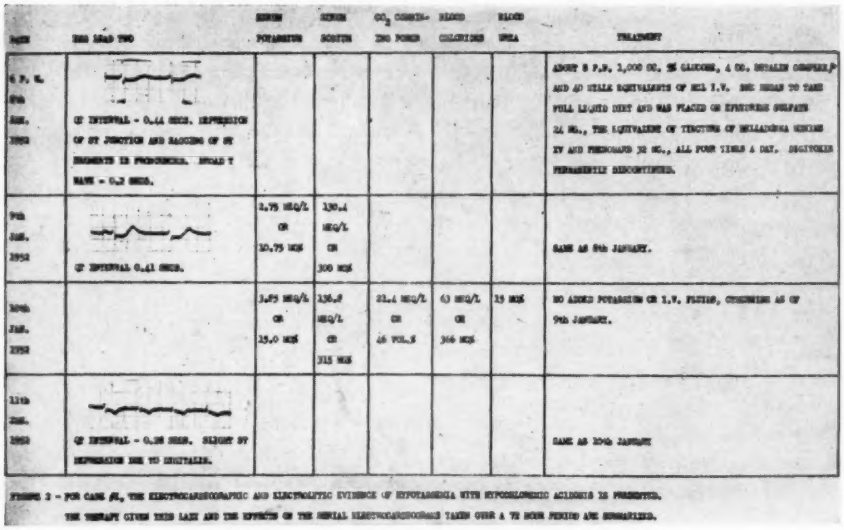

Fig. 2

time she had discontinued her ephedrine and reduced her belladonna to the equivalent of thirty-two drops per day. In June, her resting blood pressure was 150/90, and massage of the carotid sinuses, performed on both occasions, resulted only in a mild transient drop in blood pressure and decrease in pulse rate without syncope or evidence of arrest of the SA node. See figure 3 comparing her tracing taken on 12th June with that of 6th January 1952.

Case 2. J.E.Q., a 62 year old white day laborer, was hospitalized on 5th February, 1945, because of "weak, dizzy spells", occurring two or three times a week during the previous two years. Pressure on either side of his neck seemed to reproduce his symptoms which rest in a reclining position relieved. Though he had felt as though he might pass out, there was no actual history of convulsions or loss of consciousness. Physical examination revealed that massage of the right carotid sinus produced cardiac asystole for a period of 3.2 seconds. See Figure 4 for electrocardiographic verification. Except for moderate enlargement of the lateral lobe of the pros-

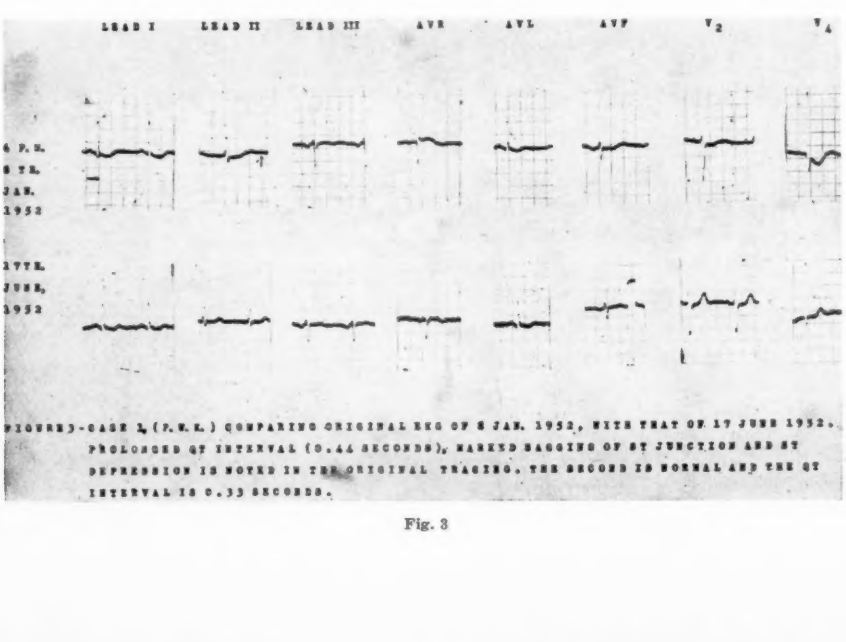

Fig. 3

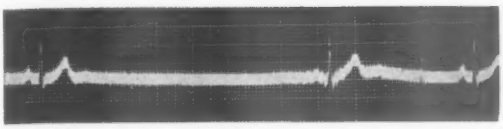

Fig. 4—Carotid sinus activity (vagal type), with S. A. node arrest of 3.2 seconds resulting from massage of right carotid sinus. (Patient J.E.Q., University of Virginia Hospital #210-301.)

tate, the rest of the physical examination was not remarkable. Tincture of belladonna, twenty drops t.i.d., and ephedrine, grains three-eighths, b.i.d., eliminated clinical and electrocardiographic evidence of his hyperactive carotid sinus.

In Table 1, the age, sex, duration of symptoms, treatment, and response to therapy are summarized for the six cases included in this study. In five out of six patients, their symptoms had been present from four months to fifteen years. Since the diagnosis of carotid sinus syndrome can readily be made, it should be considered in all cases of unexplained weakness, giddiness, or fainting. Though predominately occurring in persons over thirty-five, youth is not exempt. The concurrence of idiopathic epilepsy and an overactive carotid sinus is extremely rare. Nevertheless, older persons with cerebral degenerative disease may occasionally also be found to have hyperactivity of the carotid sinus. Such was the case in the second subject, H.B.F., in this series.

Drug therapy is summarized in Table 1. Glaucoma or allergy to atropine, belladonna, or banthine may be an absolute contraindication in the vagal type. Small amounts of sedation, phenobarb, etc., may be requisite to counteract the side effect of ephedrine, paredrine, or similar medications employed to prevent hypotension. Mechanical sources of pressure should be eliminated; if intrinsic, i.e., calcified or enlarged nodes, such should be alleviated by appropriate means; if extrinsic, stiff collars, chokers, etc., their use should be scrupulously avoided. Digitalis and hypoglycemia due either to insulin or functional derangement of the autonomic nervous system may sensitize the carotid sinus.[3,5,6,7] Except for severe heart failure, the digitalis group of drugs are con-

SIX CASES OF THE HYPERACTIVE CAROTID SINUS

Case Number	Age and Sex	Duration of Symptoms	Type	Verification	Treatment	Result
1. (P.W.K.)	65 Female	Six to eight months	Depressor primarily and vagal secondarily	Drop in blood pressure, changes in pulse and cardiac pacemaker verified by EKG.	Ephedrine sulfate 24 mg. and the equivalent of 15 drops of tincture belladonna, both four times daily.	Excellent
2. (H.B.F.)	60 Male	15 years	Vagal	Clinical and EKG evidence of cardiac arrest	*Phenobarb. 16 mg. 4 times a day.	Some improvement
3. (J.E.Q.)	62 Male	2 years	Vagal	Clinical and EKG evidence of cardiac arrest	Tincture of belladonna 20 drops three times daily. Ephedrine sulfate 24 mg. three times daily P. R. N.	Excellent
4. (B.J.J.)	60 Female	1 month	Vagal	Clinical and EKG evidence of cardiac arrest	Tincture of belladonna 15 drops, three times a day.	Unknown. No follow up
5. (C.L.R.)	59 Male	4 to 5 years	Vagal	Clinical with asystole on palpating neck.	Atropine by hypo. 0.86 mg., and then 0.43 mg. every 6 hrs. for 5 doses.	Unknown. Patient had advanced carcinomatosis
6. (G.T.F.)	69 Male	4 months	Vagal	EKG showing cardiac arrest.	Ephedrine sulfate 24 mg. daily and atropinization P. R. N.	Probably did improve but follow up not adequate

*Convulsive seizures possibly associated with cerebral degenerative disease due to cerebral arteriosclerosis.

TABLE I

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traindicated. Functional fluctuations of the blood sugar can be avoided by restricting the carbohydrate intake and increasing the amount of protein ingested at meals. If medical therapy does not control the sensitive carotid sinus, surgical denervation is indicated.

One other complication of increased activity of the carotid sinus deserves attention, namely: the occurrence of cardiac arrest during anesthesia. This dramatic and too often fatal complication is estimated to occur once during the life time of an active surgeon or about once in every ten thousand operations. Two factors which play a role in cardiac arrest are: (1) Manipulation of the neck and head with inadvertent massage of the carotid area particularly during the induction phase of anesthesia.[3,8] (2) Certain types of anesthetics have been reported to sensitize the carotid sinus.[8]. Nitrous oxide and sodium pentothal are under scrutiny for this reason. Care on the part of the anesthetist in avoiding the carotid area would remedy the first. Increasing the pre-operative dose of atropine to one or two mgm. would do a great deal to alleviate the second.

Conclusion: Hyperactivity of the carotid sinus is readily diagnosed by any alert physician. It should be considered in any case of unexplained syncope, giddiness, or convulsive seizures. Treatment as outlined is usually quite gratifying. Cardiac arrest during anesthesia, though a relatively rare occurrence, might in many cases be avoided by increasing the pre-operative dose of atropine and by the anesthetist being careful not to inadvertly massage the carotid sinus.

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Keep Plugging "Your Doctor" Film.

Local medical societies can improve their "public relations rating" in the community by booking the "Your Doctor" film for non-theatrical showings at society meetings or to PTAs, schools, churches and other groups interested in health. Sixteen millimeter prints of this 15-minute sound film now are available on loan from Modern Talking Picture Service, Inc., 45 Rockefeller Plaza, New York 20, N.Y. Produced as a documentary, this film points up ways in which the medical profession has helped to bring better medical care to everyone. During 1952 more than 12 million Americans viewed the movie in over 5,000 commercial theaters throughout the country. It is hoped that medical societies will continue to promote the film locally.

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TUBERCULOSIS IN INFANCY AND CHILDHOOD*

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Richmond, Virginia

Tuberculous infection in infancy and early childhood is more common than is generally believed. For example, in our own BCG program at the Medical College of Virginia in which clinic patients under the age of 6 months are vaccinated against tuberculosis, it has been found that about 7 per cent of the control group are already infected with tuberculosis. All except one of these children were found to be tuberculin positive when under 2 years of age.

In order to properly evaluate childhood tuberculosis, one must be aware of the pathogenesis. An adaptation of Wallgren's[1] presentation states that, following tuberculosis infection, which in over 90 per cent of the instances is in the lung parenchyma, there is a 2 to 8 week incubation period, during which tubercle bacilli are conveyed from the portal of entry by way of the lymphatics to the regional lymph nodes. The glands become hyperemic and edematous and may contain areas of caseation. At the close of the incubation period allergy sets in, as manifested by the positive tuberculin test. Near the close of the incubation period there is often a transient bacillemia and tubercle bacilli are disseminated into the blood stream and set up depots or foci.

Brailey's[2] work has indicated that (1) those children infected with tuberculosis under the age of 3 years bear a more serious prognosis than those infected after that time; (2) colored children infected with tuberculosis have a worse prognosis than do white; (3) those children who show a parenchymal lesion at the time of the first chest x-ray (after demonstration of a positive tuberculin test) bear a more serious prognosis than do those who show no parenchymal lesion, and (4) the first year following the demonstration of the presence of tuberculous infection is most important. Those children who appear to be well at the end of this time have a good chance for complete recovery.

Diagnosis of tuberculosis in childhood is effected by means of the tuberculin test.[3] This test should be performed routinely on all infants between the age of 12 and 18 months and on all new patients more than a few months of age. Since the vast majority of children with tuberculous infection exhibit no physical or roentgenological evidence of the disease, the routine tuberculin test is the only way to bring about early diagnosis in most instances. Whenever an adult is found to have active tuberculous disease, it is, of course, necessary that a tuberculin test be performed on all children who have been in contact with the diseased individual.

The routine tuberculin test may be performed with the patch test or by the intradermal use of old tuberculin solution, or P.P.D. When the patch test is used the skin is thoroughly cleansed with acetone or ether and the patch applied between the scapulae. The parent is cautioned not to allow the patch to become wet and to remove the patch 48 hours after it has been applied. Reading is done 48 hours later and there must be either marked redness and induration (0.5 cm. in diameter) or vesiculation of either or both of the top and bottom squares in order to be called positive. If there is any doubt, e.g., if there is some induration and redness of the above mentioned squares with no demonstrable vesiculation, it would seem wise to follow up with an intradermal test.

For routine intradermal use, one may choose either old tuberculin solution (0.1 mg.) or P.P.D. solution (1st strength). Reading is done 48 hours after the test is performed and the area of redness and induration must be at least 0.5 cm. in diameter in order to be adjudged positive. In reading the test in the deeply pigmented skin of Negroes, one must rely largely on the area of induration.

If tuberculosis is suspected, one must either use the patch test or begin with an intradermal test of a much higher dilution (lower strength) of old tuberculin.

Whenever routine skin testing reveals tuberculous infection in a child all adult contacts must have a chest x-ray. The routine tuberculin test in children has been found to be a definite aid in adult tuberculosis case finding.[4]

When tuberculous infection is diagnosed in a child,

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*Read before the annual meeting of The Medical Society of Virginia, at Richmond, September 28-October 1, 1952.

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prompt search for and removal of the adult contact is the first requisite for satisfactory treatment. The next requirement is careful study of the infected child with subsequent close follow-up. The child who has a positive tuberculin test, but who has no other demonstable sign of the disease either on physical examination or roentgenogram, must be classed in the same group as those which show roentgenographic evidence of mediastinal tuberculosis, with or without a demonstrable primary parenchymal focus. Adequate diet, added vitamins and plenty of rest are necessary. Further, these children must be rechecked at frequent intervals during the first few months after the disease has been diagnosed and at increasingly longer intervals thereafter. For example, a 2 year old child with mediastinal tuberculosis should have either fluoroscopic or roentgenographic chest examinations every month for the first 3 months, then again after 3 months and 6 months. After 1 year has passed an annual chest x-ray will be sufficient unless there is clinical evidence of activation of the disease process.

The child who has so-called "progessive primary tuberculosis" should have a therapeutic trial with isonicotinic acid hydrazide. If this is unsuccessful, streptomycin may be used, although most clinicians prefer to save the latter for such complications as miliary tuberculosis and tuberculous miningitis.

Today some cases of tuberculous meningitis apparently can be cured. At the Medical College of Virginia the first case of tuberculous meningitis treated with streptomycin was admitted in October, 1947. Of the first twenty cases[5] treated with streptomycin and promizole, 9 are living more than two and one-half years later. There have been many more cases since that time, of course, but there has been no significant change in therapeutic results.

Our own schedule of therapy, a modification of that suggested by Lincoln,[6,7,8] is as follows.

(1) 40 intrathecal injections of streptomycin, 25 to 100 mg. each, over a 9 week period.

(2) Intramuscular streptomycin, 20 to 40 mg. per pound of body weight per day over a 6 months period.

(3) Promizole in quantity sufficient to maintain a blood level of 1 to 3 mg. per cent over a 3 year period.

We have made many modifications of the above schedule. Para-amino-salicylic acid has sometimes been substituted for promizole and now we are adding isonicotinic acid hydrazide to our routine treatment.

In closing, then, it is our feeling that the approach to tuberculosis in childhood is threefold: (1) case finding, with removal of the adult contact and careful follow-up of the infected child; (2) prompt and judicious use of the accepted chemotherapeutic agents in the treatment of the severe forms of the disease, and (3) a BCG program in the infants of population groups with high tuberculosis morbidity and mortality rates.

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414 West Franklin Street.

PATHOLOGICAL PHYSIOLOGY OF CHRONIC LUNG DISEASE

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Introduction

The purpose of this report is to describe briefly the abnormalities in respiratory physiology that accompany chronic lung disease. Pulmonary emphysema and pulmonary fibrosis are the usual causes. For that reason, this discussion will be limited to these two conditions, and to the pulmonary hypertension that often accompanies them. There are other important and interesting conditions, but space does not permit their consideration. A short description of normal lung functions will serve to introduce the subject. There are no original observations in this report. Most of the material covered can be found in reports by the Cournand and Richards group[1,3] and by Wright and Filley[2]. Many of the physiological observations have been repeated in the Cardio-Pulmonary Laboratory at the University of Virginia Hospital.

It is the purpose of the respiratory apparatus to introduce oxygen into, and remove carbon dioxide from the blood. In doing this, it also helps to regulate the acidity of the body fluids. Lung function may be divided into two main parts: Ventilatory Function and Respiratory Function.

Ventilation includes all processes concerned with the transport of air through the respiratory passages and its distribution to the alveoli. It requires an intact thoracic cage and diaphragm with adequate respiratory muscles, patent airway, and reasonable lung elasticity and volume. Respiratory function includes the processes concerned with the passage of oxygen and carbon dioxide across the alveolar-capillary membrane. It requires a permeable membrane as well as adequate pulmonary circulation.

Pulmonary Emphysema

The disability of pulmonary emphysema can be explained by the physical, pathological and physiological abnormalities found in this disease. The most obvious abnormality is the over inflation of the lungs. Inspection reveals the relatively immobile chest held in the inspiratory position. Fluoroscopy shows the depressed and poorly moving diaphragm. The decrease in pulmonary markings as seen in the chest x-ray is further evidence of abnormal lung distention.

The excessive lung inflation is due to two factors. First, there is the loss of the pulmonary elastic tissue that normally causes contraction of the lung. The second factor is the narrowing of the lumen of the smaller bronchi and bronchioles. The presence of this bronchial narrowing may be hard to demonstrate. Wheezing, when present, indicates partial bronchial obstruction. The very prolonged expiration time so characteristic of emphysema must be due to narrowing of the bronchial lumina. Finally, the powerful effort required by the patient to force air out of the emphysematous lung must be due to obstruction.

Poor respiratory mechanics further impair ventilation. The chest that is held permanently in an inspiratory position with a diaphragm that is permanently depressed cannot function well as a bellows.

The physiological effects of these changes are important. The vital capacity is reduced. The amount of air that can be moved in and out of the lungs per minute, that is, the maximum breathing capacity, is reduced. The amount of air remaining in the lung at the end of expiration, that is, residual air, is increased.

The bronchial narrowing and loss of elastic tissue are not uniform throughout the lung. The result is that there is unequal distribution of the inspired gases within the lung. There is poor ventilation of some areas, and good ventilation of others. If the poorly ventilated areas are well perfused with blood, and this seems to happen at times, this blood will come through poorly aerated. If the ventilatory deficiency and the imbalance between alveolar ventilation and perfusion are well developed, the arterial blood will have less oxygen and more carbon dioxide than normal. If there is much retention of carbon dioxide, respiratory acidosis will develop.

Pulmonary Fibrosis

The physiological abnormalities associated with

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Read as a part of the Symposium on Chest Conditions before the annual meeting of The Medical Society of Virginia at Richmond, September 28-October 1, 1952.

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the proliferation of fibrous connective tissue, within the lung will depend on its location, amount, and histological characteristics. If the fibrosis is localized, there may be no demonstrable physiological changes. If, on the other hand, it is extensive, or, as so often happens, the fibrosis is complicated by emphysema, the physiological changes may be profound.

Fibrosis may alter pulmonary function in the following ways:

1. It may destroy or replace functioning lung tissue. Its very existence means that a certain amount of lung tissue must be lost.
2. It restricts motion of the lungs. Not only may there be large blocks of fibrous tissue that cannot expand, but, if there are strands of fibrosis running through the lung, they may restrict expansion of normal alveoli.
3. Growth of fibrous tissue within the lumen of the bronchi or its growth and contraction in bronchial walls and adjacent tissue will cause obstruction and interfere with air flow.
4. The alveolar walls may be thickened so that there is increased resistance to the flow of oxygen and carbon dioxide between the interior of the alveoli and the pulmonary capillaries.
5. In addition to these rather clear cut factors, Dr. Wright has discussed the importance of abnormal stimuli arising from the diseased tissues of the respiratory apparatus. In the normal lung there are specialized nerve endings that are sensitive to changes of tension in the surrounding tissue. They initiate reflexes that control respiratory rate and depth. It seems likely that the abnormal elasticity of the scarred lung would set up abnormal reflexes. These may explain the abnormal breathing patterns, such as excessive hyperventilation from minor exercise, that sometimes occur.

The degree of anoxemia resulting from the structural changes will depend to a large extent on the amount of fibrosis. It will also depend on the amount of blood flowing through the fibrotic areas. If the blood vessels through the diseased tissue are obliterated by the process or obstructed by vasospasm, all the blood will go through good lung tissue and there will be little or no decrease in arterial oxygen saturation. If, however, the vessels through the diseased and non-functioning lung remain open, the perfused blood will not be well aerated and arterial oxygen levels will be low. Fortunately, the blood vessels through areas of fibrosis are usually obstructed.

The abnormalities found in pulmonary fibrosis are likely to cause a reduction in the vital capacity, a decrease in maximum breathing capacity, a reduction in arterial oxygen, saturation, and abnormal breathing patterns.

CARDIAC COMPLICATIONS

Pulmonary hypertension often accompanies chronic lung disease. In emphysema the chief cause is chronic anoxemia while in pulmonary fibrosis the chief cause is obliteration of the pulmonary vascular bed.

The effects of anoxemia itself are remarkable. It causes local vasoconstriction of the pulmonary arteries, and an increase in the cardiac out-put. When chronic, it causes polycythemia and an increase in blood volume, leading to engorgement of the pulmonary vessels. The result is an increased amount of blood flowing through vessels already filled to the limit of their distensibility. The pulmonary blood pressure has to rise.

Sustained pulmonary hypertension leads to hypertrophy and finally dilatation and failure of the right ventricle. That is, cor pulmonale develops. The clinical implications are great. The patient now has the manifestations of heart failure as well as those of his original pulmonary disease.

Alterations in function that may be found with cor pulmonale include pulmonary hypertension, normal, decreased or increased cardiac output, increased venous pressure, and increased circulation time.

SUMMARY

Pulmonary emphysema and pulmonary fibrosis lead to serious alterations in respiratory function. Pulmonary ventilation is decreased. Inspired air is poorly mixed and poorly distributed to functioning alveoli. There may be defective perfusion through the alveolar-capillary membrane. As a result of these abnormalities there may be deficient oxygen and excess carbon dioxide in arterial blood. Chronic anoxemia plus obliteration of the pulmonary vascular bed leads to pulmonary hypertension and cor pulmonale.

The dyspnea of chronic lung disease is largely due to the increased respiratory effort needed to overcome

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the partial bronchial obstruction, loss of lung elasticity, and poor respiratory mechanics.

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Report Brain Operation Aids Chronically Mentally Ill.

Bilateral prefrontal lobotomy is capable of aiding the return of chronically mentally ill patients to the community, it was reported in the January 17 J.A.M.A., by Dr. Milton Greenblatt, E. Emily Robertson, M.S., and Dr. Harry C. Solomon, Boston, following a five-year study of the first 100 patients on whom the operation was performed at the Boston Psychopathic Hospital. The authors are associated with the hospital, and with the department of psychiatry, Harvard Medical School.

Bilateral prefrontal lobotomy is an operation which severs nerve fibers of the front part of the brain to keep certain nerve impulses from reaching the brain. At the end of five years, 40 of the 100 patients were residing in the community, 45 were in hospitals, 12 had died, and three could not be traced, according to the authors. Only two of the deaths were related to the operation, they added.

Twenty-nine per cent of the patients on whom information was available were considered to be making good work adjustment at the end of the five-year period, as compared to two per cent prior to the operation. This meant that they were earning a salary and that their performance satisfied their employers if engaged in labor; if engaged as housewives, they were working full time and satisfactorily, as far as their relatives were concerned.

Thirty-four per cent of the patients were considered by the doctors to have made fair adjustments following the operation. This consisted of partial remission of most if not all symptoms, or complete remission of at least a few important symptoms. The condition of 37 per cent of the patients remained essentially unchanged.

The authors stated: "The results indicate that bilateral prefrontal lobotomy is capable of effecting a rise in the level of adaptation of chronically mentally ill patients and that this rise is generally sustained over a five year period."

"Improvement in these patients is striking when their condition is compared to their preoperative state of deterioration; however, in most cases, improvement is not sufficient to reach the preillness level of adaptation."

They said that convulsive seizures occurred in 12 per cent of the patients, but that these were usually few in number and readily controlled by anticonvulsive therapy.

The operations were performed between October, 1943, and April, 1946. All the patients had suffered from chronic mental illness for an average duration of six years; the prognosis of the disease was considered hopeless. Most of the patients were skilled laborers, clerical employees and housekeepers; about half had graduated from high school.

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SPONTANEOUS HEMOPNEUMOTHORAX OCCURRING IN A FEMALE

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A sizable literature on spontaneous hemopneumothorax has accumulated despite its infrequent occurrence. The rarity, difficult problems encountered clinically and changing concepts of therapy, have all contributed towards this interest. Up to 1951, there were 62 cases of this entity described in the literature. In two of these cases the patients were females. It is the purpose of this paper to describe the third case of spontaneous hemopneumothorax occurring in a female, and to comment upon the interesting aspects of this condition.

Spontaneous hemopneumothorax was first reported by Pitt[1] in 1900. Up to 1942, Hartzell[2], in a review of the literature, collected 40 cases and added 3 of his own. In 1951, Moser[3] reported 4 cases and noted that there were 58 cases recorded up to that time.

Spontaneous hemopneumothorax is a complication of spontaneous pneumothorax and is best understood by considering the pathogenesis of the parent condition. The evolution of the latter entity is still in dispute though several theories have been advocated. There is general agreement that spontaneous pneumothorax is caused by rupture of an emphysematous bleb, but where such authors as Macklin[4] and Ornstein[5] differ, is on the formation of the bleb itself.

In animal experiments Macklin[4] attempted to prove that the precursor to spontaneous pneumothorax was air dissecting along the pulmonary interstitial tissues subpleurally. Ornstein[5], on the other hand, ascribed bleb formation to a check valve mechanism in the distal bronchioles. The complication of hemothorax is fortuitous and depends on the presence of a blood vessel either in the bleb, or in an adjacent pleural adhesion. The extent of the hemopneumothorax then depends on the size of the bleb and the elasticity of the adjacent lung tissue.

CASE REPORT

This 24 year old white single sales girl was entirely well until October 17, 1951, when she noted aching precordial and left lateral chest pain. This pain was fleeting for the first three days, and then became severe and persistent. There was no cough, fever or antecedent illness. At onset of the illness and for 2 days thereafter only a rectal temperature of 100° and a slightly injected pharynx were noted on physical examination. On the third day, the patient became acutely ill, moderately dyspneic, a friction rub was heard at Erb's point and there was splinting of the left chest. Later that day there was cough, expectoration of thick yellow sputum and left pleural effusion extending up to the 8th rib posteriorly. As the effusion extended, the pain subsided and the pulse rate increased to 120. At no time was the blood pressure recorded below 110/70. Hyperresonance, absent breath sounds and a succussion splash were then noted. Prior to hospitalization the patient was placed on antibiotics and Demerol for relief of pain. The patient was hospitalized one week after onset

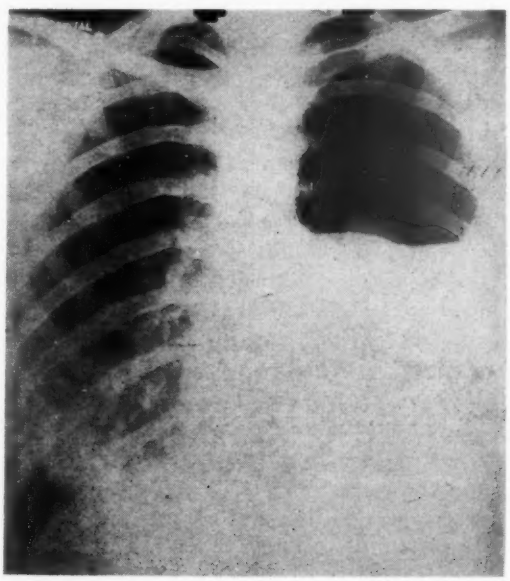

Fig. 1. The initial chest film reveals a fluid level in the left pleural cavity extending up to the 6th posterior rib. The small density above the fluid (blood) is a portion of the collapsed left lung.

of her illness. There, the physical findings were as above; left pleural effusion now up to the 6th rib posteriorly with air above the fluid, tachycardia and B. P. 117/70. Pertinent laboratory findings were: Hb of 50%, RBC 2.4 million, WBC 8,800 with 81% polys. The initial X-ray (Fig. 1) revealed pleural fluid up to the 6th rib posteriorly, a collapsed left lung and air above the fluid. The right lung was normal. Thoracentesis was performed the day following hospitalization and 800 cc. of blood were removed from the left pleural cavity with appreciable relief of dyspnea. A 500 cc. blood transfusion was given at this time. The following day 900 cc. of blood were removed from the left chest. The dyspnea then disappeared. X-ray of the chest after aspiration of 1700 cc. of blood (Fig. 2) revealed a

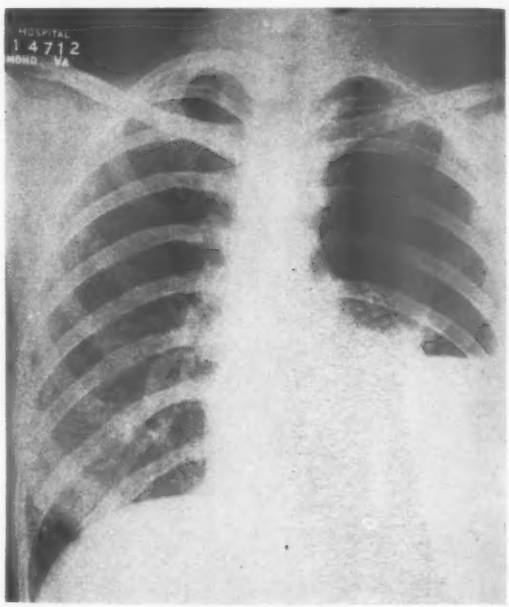

Fig. 2. A considerable re-expansion of the left lung is seen after the removal of 1700 c.c. from the left pleural cavity. The fluid level is now at the 8th posterior rib.

decrease in the fluid level and some expansion of the collapsed lung. The patient's subsequent course in the hospital was uphill. In all, 4 thoracenteses were performed with the removal of 2600 cc. of blood from the left pleural cavity. X-ray of the chest at this time (Fig. 3) revealed almost complete expansion of the left lung, and blunting of the left costophrenic angle. At no time was the patient's temperature above 100 degrees. Her blood pressure

Fig. 3. Complete expansion of the left lung is seen after removal of 2600 c.c. of blood from the left pleural cavity.

remained stable throughout. By the end of her hospital stay and following two blood transfusions the Hb was 88%, RBC 4.5 millions. The duration of her illness was six weeks in all. Since her return home she has gone back to her job as sales girl. A follow-up chest film on December 15 revealed only blunting at the left costophrenic angle.

DISCUSSION

The onset of spontaneous hemopneumothorax is not necessarily a sudden one, as is generally believed. Leach,[6] in his review of 126 cases of spontaneous pneumothorax, noted an insidious onset in 35 of his cases. This is in keeping with the events which took place in our case. The patient had malaise for 3-days, then noted increasing degrees of left lateral chest pain, and finally with probable diaphragmatic involvement, left shoulder and scapular pain.

The majority of the cases in the literature, in approximately 80%, are on the right side. In our case the episode occurred on the left side. There has been no adequate explanation for the predilection of this entity for the right pleural cavity. Nor has any reason been put forth as to the predominance of males with this condition. The patient with spontaneous pneumothorax is usually a healthy robust

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individual. On the other hand, spontaneous hemopneumothorax occurs in an individual who is healthy, but somewhat underweight. It is the accepted view, that there is no history of previous disease or trauma. When hemopneumothorax occurs in an individual with tuberculosis, the episode is not called spontaneous, and there is usually widespread evidence of tuberculous pulmonary involvement.

In our case, the purulent sputum was secondary to a superimposed infection in the collapsed lung. By the time the patient was hospitalized, the sputum became mucoid and culture of both sputum and pleural blood were negative.

We believe that this patient did so well because of prompt pleural aspiration, replacement of whole blood, and the liberal use of antibiotics. Until very recently these concepts were much disputed in the literature. Certain authors have been opposed to prompt and rapid removal of blood from the pleural space. Their arguments have been that the production of a negative pleural pressure would stimulate increasing hemorrhage. Furthermore, there have been some advocates of cautious removal of blood and replacement with air into the pleural cavity. Recent observations by Johnson[7] have demonstrated that prompt aspiration of all possible blood consistent with the welfare of the patient, is the treatment of choice. There is usually no difficulty in the thoracentesis of blood because, as Cosgriff[8] has demonstrated, it has lost its coagulability. This absence of clotting is believed to occur approximately two hours after the onset of the hemopneumothorax, and is most likely due to the whipping action of the diaphragm, mediastinal shift and the resultant deposition of fibrin on the pleural layers.

Replacement of blood volume has been interdicted by Head[9] because of the possibility of increased blood pressure with disturbance of clot formation at the bleeding site. This has not been the experience of Johnson[7], and was not true in the case being reported.

Until recently, empyema and subsequent surgery in spontaneous hemopneumothorax was a common occurrence. This was true in almost one-half of the cases reported. With the advent of antibiotics, if the entity is recognized in time, this complication can be avoided. The liberal use of chemotherapy is advocated because of the ability of blood to act as an excellent bacterial culture medium. In our case 500 grams of chloroamphenicol were used.

The drug of choice for control of pain is, in our opinion, Demerol. The excessive suppression of the cough reflex and depression of the respiratory center can thus be avoided.

It is important that the diagnosis of spontaneous hemopneumothorax be made as early as possible and treatment as outlined be instituted. This can not be emphasized too strongly. The diagnosis is not difficult if the clinical entity is kept in mind. Certainly the simple procedure of shaking the patient and eliciting the succussion splash, will make the diagnosis in a young healthy adult male, and rarely, adult female. The later complications of empyema and fibrothorax can be avoided by prompt and vigorous treatment.

CONCLUSION

A case of spontaneous hemopneumothorax in a young previously healthy female is reported.

The literature and interesting aspects of this clinical entity have been commented upon.

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Medical Society of Virginia Cancer Committee

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Reprints of this and preceding Bulletins may be obtained from this office

MARCH 1, 1953

BULLETIN

Carcinoma of the uterus has been the subject of many of these Bulletins. Your Committee has cited instances in which disease has been allowed to progress without treatment or with incorrect treatment because standard diagnostic procedures were omitted and because treatment was carried out before uterine pathology was accurately estimated. The other side of the picture has also been presented — instances in which uterine carcinoma was diagnosed and treated in an early curable stage because a physician was alert and thorough.

Our readers will then be particularly interested in an article by Dr. James C. Doyle of the University of Southern California Medical School, in the January 31, 1953, issue of the J. A. M. A. The title is "Unnecessary Hysterectomies" and the report consists of a statistical analysis of 6,248 hysterectomies performed in 35 hospitals during 1948. The following highlights are especially relevant to the problem posed by carcinoma of the uterus.

Gynecologists now teach that when hysterectomy is indicated, total removal should be done if practical. However, in Dr. Doyles' unprecedentedly extensive sampling, a subtotal hysterectomy was performed in 50.5 per cent of the cases. In patients under 50 years of age, subtotal removal was chosen in 55 per cent of cases; in patients over 50, the figure dropped to 33 per cent. The sharp difference in the two age groups probably reflects a healthy awareness of the malignant potentiality of the cervix, but the overall figure indicates a disappointing lag between advances in medical teaching and advances in medical practice. Of the 6,248 hysterectomies,

35.7% were performed by general surgeons of which 48.5% were subtotal

33.0% were performed by general practitioners of which 70.8% were subtotal

31.3% were performed by gynecologists of which 31.1% were subtotal

Analysis of the patients' recorded symptomatology resulted in some startling figures. In 340 women, or 5.4 per cent, there was no symptomatology, and 99 of these patients were under 40 years of age. Furthermore, in 548 patients with no symptoms or with only such complaints as headache, dizziness, palpitation, and nervousness, pathological study of the removed uterus revealed no lesion nor was any indication for hysterectomy recorded. To quote Dr. Doyle, "The low incidence of some of the complaints that commonly accompany lesions warranting hysterectomy is remarkable." For instance, the percentage of the patients complaining of backache was only 11.3 per cent; of vaginal discharge, 6.9 per cent; of postmenopausal bleeding, 5.2 per cent; of dyspareunia, 1.8 per cent.

In 160 cases, no preoperative diagnosis was recorded. In 130 cases, the preoperative diagnosis was retroposition of the uterus, which alone does not justify hysterectomy; and in this group, not one instance of pathological abnormality was discovered.

In 584 cases in which chronic cervicitis was the sole or one of the preoperative diagnoses, cervical biopsy was not done in 476 or 81.5 per cent.

Menstrual aberrations were reported by 4,459 women, of whom 3,841 or 86 per cent were not given the benefit of a diagnostic curettage.

Conditions missed preoperatively included 45 cases of carcinoma of the fundus and 20 of carcinoma of the cervix. Not one of these 65 women had had a Papanicolaou study, a cervical biopsy, or a curettage.

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In fact, of the 6,248 women subjected to hysterectomy, only 618 or 9.8 per cent had had a diagnostic curettage and only 109 or 1.7 per cent had had a cervical biopsy.

Dr. Doyle concluded that, for one reason or another, 2,458 or 39.3 per cent of the hysterectomies were open to criticism while 3,790 or 60.7 per cent seemed justified.

Perhaps the most revealing figures compiled by Dr. Doyle are those demonstrating a striking consistency in the level of physician performance in each hospital staff, regardless of the criterion selected for comparison. The following examples are illustrative:

	Best Hospital	*Worst Hospital*
Hysterectomies open to criticism	5.2%	83.7%
Hysterectomies performed in absence of lesions or relaxations	1.7%	33.1%
Diagnosis contraindicated hysterectomy	0. %	3.0%
Preoperative diagnosis confirmed	78.9%	45.7%
Diagnostic curettage prior to hysterectomy	59.6%	7.2%

The tremendous value of and real need for this sort of self-examination is clear. Deficiencies faced and defined can be corrected.

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TRICHINOSIS
A CASE REPORT*

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The incidence of trichinella spiralis infestation seems to be decreasing, as would be indicated by a report from San Francisco[1] that 24% in 1936 and 8% in 1949-50 of diaphragms of unselected autopsy material contained parasites. However, many of these cases were unrecognized and untreated during life, and for that reason it remains important that one be familiar with the clinical features of trichinosis, even though at times bizarre, in order that the diagnosis may be established, therapy begun, and preventive measures undertaken.

The following patient was seen recently at the Chesapeake and Ohio Hospital, and is believed to represent a rather typical acute, moderately severe, but uncomplicated case of trichinosis.

CASE REPORT

Mrs. G. T., a 25 year old white German war bride, having lived in this country three years, was admitted to the hospital on February 10, 1952, complaining of "aching all over."

The patient had been well until 12 to 14 days before admission when she noticed the onset of generalized malaise, nausea, abdominal cramps, and watery diarrhea. She was treated with "sulfa" drugs and penicillin without improvement. The diarrhea subsided 4 days prior to admission, but the following day chilly sensations, fever, and swelling around the eyes appeared. Two days prior to admission the patient noticed aching in her shoulders, forearms, and calves. The systems review was negative except for amenorrhea of approximately two months duration.

Physical examination revealed a well developed and well nourished female with an oral temperature of 100°F., pulse 74, respiration 20, and blood pressure 110/70. There was minimal periorbital edema, and flushing of the face, and several small bilateral cervical nodes were palpable. Marked tenderness of both deltoid regions, forearms, and calves was present, as was tenderness of the lower abdomen. Pelvic examination revealed a soft uterus of twice normal size.

The initial impression was trichinosis with pregnancy. The patient was questioned further with particular emphasis on possible ingestion of raw or poorly cooked pork products. She stated that she frequently ate raw hamburger sandwiches, and had done so within the week preceding onset of symptoms.

Laboratory studies revealed a normal hemoglobin, urinalysis, stool and serology. The white count was 21,400, and the differential count revealed 7% lymphocytes, 9% monocytes, 72% neutrophil segmented cells, and 12% eosinophils. Blood urea, sugar, sedimentation rate, and agglutinations for typhoid, paratyphoid, and brucella abortus were normal or negative. A chest x-ray was normal, as was an electrocardiogram.

Repeated differential counts revealed eosinophils of 26%, 41%, and 25% respectively. All attempts to demonstrate trichinella spiralis larvae in the peripheral blood failed, as did attempts to demonstrate them in bone marrow and spinal fluid. A biopsy of the left deltoid muscle on the twelfth day, however, was reported to reveal several foci of inflammation containing the larvae.

The patient's hospital course was characterized by daily elevations of temperature to 101°, 103°, or 105°F., and she complained frequently of muscular aching. Medication consisted of aspirin, ten grains, four or five times daily. On the eighth hospital day the patient had a complete abortion.

On the thirteenth hospital day, the patient's muscular aching had decreased, and her daily spikes of temperature were less marked. She was discharged with instructions to restrict her activities, and to return in four days for a follow-up examination.

On return, her symptoms had subsided and she was afebrile. A trichinella skin test at that time was negative; however, five days later this was re-

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*Read before the meeting of the Medical Society of the Valley of Virginia at Winchester, Virginia, May 22, 1952.

peated and was strongly positive within twenty minutes.

The patient was contacted recently, two and one-half months after her hospitalization, and stated that she was asymptomatic and performing her usual household duties.

LIFE CYCLE OF TRICHINELLA SPIRALIS AND ASSOCIATED CLINICAL FEATURES

A knowledge of the life cycle of trichinella spiralis is necessary for an understanding of the course of the disease and for the methods of treatment and of prevention. The cycle begins with the ingestion of uncooked or poorly cooked pork or pork products containing live encysted trichinella larvae. These are liberated by protein digestion, in the small intestine, and within two to four days mature, mate, and burrow deep into the mucosa. During this phase of the cycle the patient is prone to develop many symptoms referable to the gastro-intestinal tract, depending on the length of time the adult worms remain in the intestine. This, in turn, is thought[2] to depend on (a) the state of host immunity and (b) the heaviness of the infestation.

Usually within seven days the adult female has begun discharging larvae into the thoracic duct via the lacteals, and then into the general circulation. Within the four to six weeks of life, the female may discharge from 1,000 to 1,500 larvae. The male of the species dies and is excreted.

As trichinellae circulate in the blood, all tissues may be invaded including: serous cavities, skeletal musculature, heart, lungs, gallbladder, pancreas, small bowel, peritonsillar tissue, brain and cerebrospinal fluid. It is during this phase of the cycle that one observes periorbital edema, spiking temperature, and the various symptoms referable to the particular tissue involved.

Within ten days to three weeks the larvae concentrate within the skeletal muscle, encystment begins, and the symptoms are for the most part referable to the musculoskeletal system.

DIAGNOSIS

The diagnosis of trichinosis is usually not difficult, especially when a history of ingestion of raw or poorly cooked pork, followed by gastro-intestinal complaints, periorbital edema, fever, and muscular aching is elicited. However, with or without this rather typical history, the laboratory is most helpful in confirming the diagnosis.

Eosinophilia present in 88.7% of a recent series[3] of 250 cases, is the most constant finding. The eosinophils usually range from 25% to 40%, but may reach 90% within seven to fourteen days; however, it should be noted that there is no correlation between the percentage of eosinophils and the degree of infestation,[4] and other diseases causing significant eosinophilia, such as allergies, periarteritis nodosa, and other parasitic infestations and Hodgkin's disease should be excluded.

Muscle biopsies are diagnostic when uncoiled, unencysted, or recently encysted trichinae larvae are demonstrated. This is usually possible after the tenth day of infestation. Heaviest infestation is usually found near the tendinous portions of the diaphragm, gluteus, pectoral, deltoid, gastrocnemius, or intercostal muscles, in that order of frequency.

The intradermal skin test becomes positive between the second and third week of infestation, and is very useful as a bedside test because of the fact that positive reactions occur within fifteen to twenty minutes. False positive reactions do occur, however, in a few instances.

Complement fixation, precipitin, and flocculation tests may be helpful; however, these involve specialized procedures not often available. Occasionally larvae may be demonstrated in the peripheral blood, stools, or spinal fluid, and should be searched for when practicable.

PROGNOSIS

The prognosis is good in light isolated infestations; however, in epidemics, there may be a mortality of from 5% to 30%.[5] Death, when it occurs, is usually within four to six weeks from the onset, and is due to secondary pneumonia or myocardial failure. Some people have ascribed it to "toxemia."

TREATMENT

Treatment is non-specific and confined to supportive measures and control of symptoms. Supportive measures include maintenance of hydration and proper fluid balance, particularly during the stage of gastro-intestinal involvement, and the use of antibiotics and digitalis if necessary. Analgesics, though, may be the only medication necessary.

Recently,[6] however, ACTH has been used in the

treatment of trichinosis, with beneficial effects, thought to be non-specific in nature.

PREVENTION

The most frequent and important reservoir of human infestation is the hog, which has become infested with trichinellae from feeding on garbage containing uncooked pork scraps. Rats harbor the larvae, and thus may be another source of infestation in hogs.

Prevention of human trichinosis, then, can only be accomplished by public education to the necessity of thoroughly cooking or freezing pork. Freezing at minus 15°C. for twenty days or minus 18°C. for twenty-four hours will readily kill the larvae.[7]

Animal infestation can be reduced by cooking garbage used as feed, and by proper rodent control.

DISCUSSION

The case presented represents the typical picture of trichinosis. Within a week following the ingestion of a product assumed to contain raw pork, the patient noticed symptoms of gastro-enteritis, characteristic of the stage of invasion of the intestinal mucosa by adult trichinellae. A few days later she noticed fever, periorbital edema, and generalized aching, characteristic of the stage of migration of the larvae in the blood. Spiking fever and musculo-skeletal symptoms continued for approximately one month after infestation, and gradually subsided, presumably as circulating larvae began encystment in the muscles.

Eosinophilia, up to 41%, was present during the third week, a muscle biopsy was positive during the fourth (Figure I), and a skin test became positive during the fifth week after infestation.

Treatment was entirely supportive, and the patient made an uneventful recovery within five weeks, except for having a complete abortion during the height of her illness. It is unfortunate that the fetus was not examined for the presence of trichinellae larvae; however, placental tissue examined, failed to reveal the presence of the larvae.

Fig. 1.—Recently encysted trichinella larva demonstrated in a biopsy of deltoid muscle.

SUMMARY

A proved case of trichinosis is presented. A brief resumé of the clinical features, aids in diagnosis, treatment, and prevention is outlined and discussed.

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THE EFFECTS OF INSULIN HYPOGLYCEMIA ON THE DIABETIC HEART*

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The high incidence of heart disease in the diabetic population is a well known and a well documented fact. This was clearly shown in Dr. R. L. Bailey's excellent paper[1] several years ago in which he pointed out that in a series of five hundred unselected diabetic patients thirty-five per cent could be classified as having arteriosclerotic heart disease. Other series bear out this high incidence of heart disease in diabetes. In a study[2] at Western Reserve University of two hundred and fifty diabetic patients, fifty per cent had heart disease caused by coronary arteriosclerosis and hypertension, alone or combined. Blotner[3] found in seventy-five fatal cases of diabetes mellitus autopsied at Peter Bent Brigham Hospital forty-five per cent showed marked coronary vessel disease and ten per cent had died of myocardial infarction.

Thus in a disease in which heart disease is such a frequent complication, it is of interest as to what effect the treatment of the diabetes—namely, the use of insulin—has on the heart. In reviewing the literature concerning this subject one finds that many experimental and clinical studies have been made on the effects of insulin on the normal diabetic heart and on the heart of man and animal without diabetes. Although there appears to be a general concensus of opinion that the elderly diabetic with coronary disease should be treated with caution, a careful and detailed study of the effects of insulin on the heart in a large group of elderly diabetics is wanting. Therefore, in discussing this subject one must rely a great deal on what has been learned from its effects on the normal heart and the normal diabetic heart, and fill in with the impressions and case reports that have been given concerning the effects of insulin on the diseased diabetic heart. It is hoped that more studies will be made in the future concerning this subject.

There is good evidence that insulin has no deleterious effect on the normal diabetic heart. Cruickshank[4] has shown in the isolated heart that under hyperglycemic conditions insulin increases oxidation of sugar and the deposition of glycogen in heart muscle. During severe hypoglycemia the heart glycogen is used, but still falls only to normal. If hypoglycemia persists, the heart is able to carry on and insulin continues to protect the heart.

Insulin hypoglycemia does not cause important electrocardiographic changes in the normal heart, nor does repeated periods of insulin hypoglycemia produce persistent changes in the electrocardiograph. Root,[5] in his study of young diabetics, found changes in conduction time to be very rare and ventricular extrasystoles very infrequent. T-wave changes were found, but were of only slight degree. His studies were conducted when blood sugars were elevated and low, but not when the patients were in insulin shock. Goodrich[6] has found that the most frequent electrocardiographic change in hypoglycemia to be the lengthening of the electrical systole. Otherwise, he found no important electrocardiographic changes. Middleton and Oatway[7] found in addition to decrease in amplitude of T-waves in diabetics in insulin shock that there is a tendency for P-waves to be decreased also. They found that about half of their tracings showed an increase in the auriculo-ventricular conduction time. Of course, isolated cases of various arrhythmias and perhaps sudden death can be cited in such patients, but, by and large, these are few in patients with normal hearts and should not be considered as occurring often enough to deny close control to diabetic patients without heart disease. The staying off of coronary sclerosis and the prolongation of life in the diabetic by close control with insulin certainly offsets any fear of injury that insulin may cause the normal diabetic heart.

On the other hand, the diabetic patient who has developed coronary disease should not be subjected to hypoglycemia and it is felt by many that it is safer to allow them to remain slightly hyperglycemic as insulin angina and myocardial infarction frequently occur following shifts to hypoglycemia.

The cause of insulin angina has not been explained clearly, although the electrocardiographic changes during hypoglycemia have recently been

*Presented in part at the clinical session of the Virginia Diabetes Association, Richmond, November 7, 1952.

given a reasonable explanation by Parrish and his co-workers[8]. They have postulated that the electrocardiographic changes in hypoglycemia are due to intracellular shifts of the potassium ion secondary to the effect of insulin and to epinephrine liberation. In their study of fourteen patients in whom they had induced hypoglycemia by insulin, all showed electrocardiographic changes and a fall in serum potassium levels. Electrocardiographic changes consisted of flattening of T-waves, ST segment depressions, and prolongation of Q - T intervals. The serum potassium fell an average of 1.89 meq. per liter. Those patients who received potassium chloride had a reversion of the T-waves toward normal. Priscoline was also given some of these patients and they also had a reversion of the T-waves toward normal. It was apparent in their study that the electrocardiographic changes were not necessarily due to hypoglycemia, as some of the electrocardiographic changes reverted to normal while the blood sugar level was still low. In those patients whose electrocardiograms reverted to normal with priscoline it would seem that by blocking the epinephrine with a return of the electrocardiogram to normal that the epinephrine is also a cause of the changes.

Insulin has been proven not to cause coronary constriction *per se*. On the contrary, studies[9] reveal that it causes coronary dilatation. However, some feel that the electrocardiographic changes and the clinical anginal symptoms following insulin are actually due to myocardial ischemia caused in an indirect way. It is thought that the low blood sugar causes an outpouring of epinephrine and this by causing an increased heart rate and output, the extra work brings about the myocardial ischemia. Cannon, McIver and Bliss[10] have shown in cats that if the adrenals are removed insulin hypoglycemia does not cause tachycardia. They feel that the pallor, tachycardia, pupillary dilatation and symptoms of insulin shock may be due to sympathetic stimulation. Other workers[11] have found that the circulatory changes found with insulin hypoglycemia which resemble epinephrine like signs do not occur if prostigmine has been administered. This also seems to bear out that some of the changes are due to epinephrine. It is interesting to note along this line that Harrison[12] has found in many individuals who supposedly had cardiac neuroses with symptoms of palpatation, weakness, pain in the chest, and dizziness, often have relative hypoglycemia. These are symptoms associated with the injection of epinephrine and may bear out Cannon's theory. Greene[13] in England has also made a similar report of patients who had been diagnosed as cardiac neurotics and were found to have hypoglycemia.

Soskin and Katz[14] believe that insulin causes electrocardiographic changes that are due in part to insulin *per se*, since these changes are only partially reversed by carbohydrate administration and are then not all due to the hypoglycemia. This has been borne out by the study of Parrish which has already been mentioned.

Messinger[15] has made a rather interesting study. X-rays made during and after hypoglycemic shock treatment revealed the aortic bulb and ascending aorta to be widened. This certainly might be important in causing cardiac pain as these changes occur to such a degree that signs of aortic insufficiency developed. These changes were reversible in his patients and were considered physiological changes rather than pathological.

There seems to be a variance of opinion concerning the effects of insulin on the blood pressure. Goodrich[6] found the blood pressure increased in 84% of his non-diabetic patients with insulin shock, averaging a rise of 23 mm. systolic.

Middleton, in his study of diabetic patients with insulin shock, found that the systolic blood pressure of sixty per cent of his cases fell, and seven per cent of the diastolic readings. Some of the cases of myocardial infarction which occur in elderly diabetics with insulin hypoglycemia may well be due to the fall in blood pressure with resulting coronary insufficiency.

Whatever may be the mechanism causing cardiac pain and myocardial infarcation in elderly diabetic patients receiving insulin, it certainly does occur, as has been shown by various reports. Gignon[16] as early as 1923 reported a case in which the patient died after three doses of insulin. Turner[17] in 1930 and Modern[18] in 1931 pointed out that insulin could cause cardiac pain. There are many other such reports, but, as has been stated, they are mostly isolated case histories and many of the other writers in warning against the use of insulin in elderly diabetics are impressions and are not detailed statistical

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reports. Goskin[19] felt that insulin was responsible for a number of cardiac deaths in diabetes. In his study of diabetics with evidence of coronary sclerosis or angina whose blood sugar levels were kept at a low normal level every case developed a fall in blood pressure, a rapid heart, irregularity, or paroxysmal tachycardia. The electrocardiograms usually showed an intraventicular block. On these patients intravenous glucose usually reversed these changes. He postulated that patients with arteriosclerotic heart disease required higher blood sugar levels than others. Blotner[3] gives a history of three diabetics who developed myocardial infarction which he thought resulted from insulin.

Liebow[2] recently pointed out that over-dosage of insulin may result in cardiac complications. He cited hypoglycemia as causing angina pectoris, myocardial infarction, congestive heart failure, the development of abnormal heart murmurs, and an increase in heart size. He concluded that "insulin reactions are associated with cardiac complications particularly in the older age group. Complete aglycosuric control of elderly diabetics by the use of insulin is not desirable." Duncan[20] in his book also feels the same concerning close control of the elderly diabetic. Marble,[21] however, feels that, with careful control, these patients can be kept free of glycosuria.

Smith[22] has pointed out that sudden changes in the blood sugar levels are harmful in arteriosclerotic diabetics. He cites an example of insulin angina when the blood sugar became elevated or when reduced. However, he felt that it was better to leave these patients in hyperglycemia.

Thus, it can be generally stated, in treating diabetic patients, first, that young diabetics should be controlled closely with insulin, as evidence points to no serious myocardial effects. Second, that those diabetics who do have coronary disease already present should not be allowed sudden changes in blood sugar levels, especially toward hypoglycemia.

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PUBLIC HEALTH

Mack I. Shanholtz, M.D.
State Health Commissioner of Virginia

Influenza

This year, as last, Virginia has experienced an epidemic of influenza-like infection that has been widespread over the State. Based on reports from the National Office of Vital Statistics, it is evident that the Virginia experience is part of a widespread prevalence over the United States, particularly the Eastern part. Confirmation of diagnosis of A-Prime influenza by virus isolations or serologic tests have been made in persons living or stationed in twelve Central States and four along the Eastern Seaboard.

The typical clinical picture as given by attending physicians is that of sudden onset with headache, usually frontal, and generalized body aches. There may be sore throat and usually non-productive cough. In uncomplicated cases, the respiratory symptoms are reported to be minimal. It has been pointed out that there may be two temperature patterns. After a sudden onset with high temperature, the course is usually 2-3 days; while with low fever at onset, the course is usually longer.

Although gastro-intestinal symptoms have occurred in some cases, they have not been the rule. Following a drop in temperature, most victims of the disease have felt profoundly exhausted. There has been no significant rise in the number of deaths from influenza and pneumonia.

According to reports from physicians, the antibiotics appear to be of little value at the time of onset. When the course is prolonged or a relapse has occurred, some physicians feel that the antibiotics are of value in preventing or lessening the severity of complications.

Each year at the season of high incidence of influenza-like infections, physicians have been alert to the possibility that the country might be in for another pandemic of influenza similar to that of 1918. In the comparatively mild epidemics of recent years various strains of the influenza virus have been implicated as causative factors.

Some of the suggested reasons for the mildness of recent epidemics are:

1. The possible development of a degree of population immunity by repeated contact with the infective agent
2. Milder strains of the virus
3. More effective treatment with the antibiotics.

Monthly Report of the Bureau of Communicable Disease Control

	January 1953	January 1952
Brucellosis	6	1
Diarrhea and Dysentery	201	405
Diphtheria	20	10
Hepatitis	244	55
Measles	280	944
Meningitis (Meningococcic)	29	19
Poliomyelitis	5	4
Rocky Mt. Spotted Fever	0	1
Scarlet fever	121	81
Tularemia	8	12
Typhoid and Paratyphoid	7	3
Rabies in Animals	50	48

AMA Conference on Special Health Services in Industry.

Current trends in direct service plans (health services in industry provided through salaried or panel physicians) . . . ways and means of maintaining high standards of medical service directly to certain groups of industrial workers . . . were aired at an informal conference on Direct Service Plans, by a joint committee of the AMA's Council on Medical Service and the Council on Industrial Health in January during the Annual Congress on Industrial Health.

Viewpoints of labor, management and the medical profession were expressed as more than 50 representatives of plans sponsored by labor and industry sat down with representatives of state and county medical societies. Many suggestions regarding future developments in programs designed to render medical care through voluntary methods were presented for study and future discussion.

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MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals

A Statistical Story of the Manic-Depressive Psychoses.*

Individual statistical studies of various diagnoses of mental diseases indicate that each has its own story. Some types remain fairly static as to the incidence of the disease and others vary, affected largely by situations. The story of the manic-depressive psychoses is a varying one.

The general trend of the number of admissions, those remaining in hospital and the rate to general population has been downward. There have been slight increases, which have coincided with changes in economic, war conditions, etc.

In 1943 those diagnosed as manic-depressive psychoses constituted 17.8% of all first admissions. In 1951 this had dropped to 7.2%. The readmissions followed almost the same pattern as to trend, but with a larger percentage of the readmissions being diagnosed as manic-depressive psychoses. The high point was in 1943 with 34.9% as compared with 18.8% in 1951 at the close of the period. However, the low point for this 12 year period was in 1949 with 17.7%.

The number of first admissions diagnosed as manic-depressive psychoses ranged from 347 in 1942 to 130 in 1951 and readmissions from 243 in 1942 to 206 in 1949, closing the 12 year period with 212 in 1951.

The number of manic-depressive psychoses resident in hospital diminished gradually for the 12 year period from 2,408 in 1940 to 1,789 in 1951. The percentage of this disease to all residents was 26.3% in 1940 with a gradual decline to 18.0% in 1951. This was a reduction of 31.6% for this particular disease.

The following chart gives a graphic picture of the number of admissions and resident patients during the 12 year period of 1940 through 1951.

The ratio of first admissions, diagnosed as manic-depressive psychoses, to general population reduced at a much greater rate than the actual number. The high point was in 1942 with 13.0 per 100,000 of general population and in 1951 the ratio was 3.9. This is a large decrease in the incidence of this type of mental disorder. Those resident in hospital

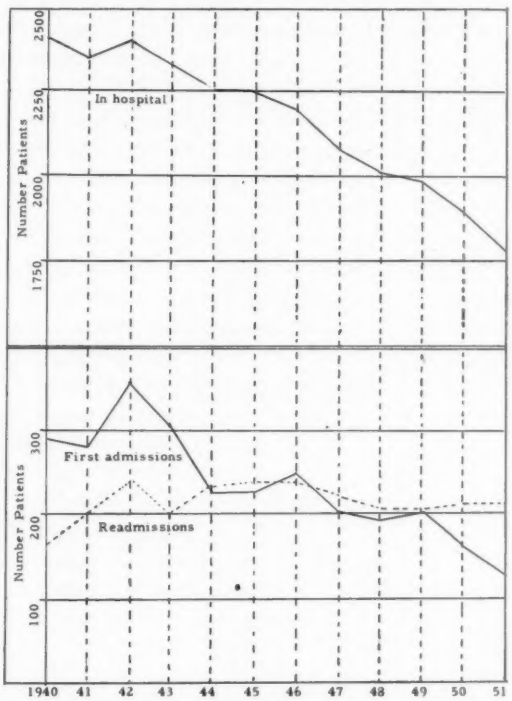

Chart No. 1. Number first and readmissions remaining in hospital for years 1940 thru 1951 of manic-depressive psychoses

showed a similar trend from 89.4 per 100,000 of general population in 1942 to 54.7 in 1951. This is a reduction of 38.8%.

In a special study in 1950 of the number of patients with manic-depressive psychoses released from the hospital within a year of their admission date, 74.6% of the first admissions had been released and 70.7% of the readmissions. The deaths were 4.4% and 3.9% respectively. Thus only 21.0% of the first admissions and 25.4% of the readmissions were still in hospital at the end of the year from their date of admission.

A similar study was made for a six months period for those admitted in the fiscal year of 1951. At

*Article by Edna M. Lantz, Statistician, Department of Mental Hygiene and Hospitals.

the end of the six months from the date of admissions, 64.9% of the first admissions had been released and 52.9% of the readmissions. Deaths were 6.1% and 4.2% respectively. This strongly suggests that a more intensive effort during the first six months would pay dividends by way of reduced hospital stay. The following chart shows this graphically.

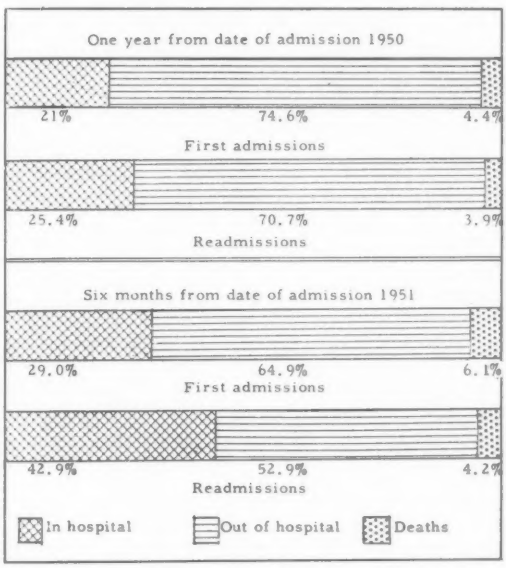

Chart No. 2. Status of First Admissions and Readmissions to State Hospitals diagnosed as manic-depressive psychoses one year from date of admission for 1950 and six months from date of admission for 1951

The average age of admission for the manic-depressive group for the 12 year period was 37.8 years and the readmissions 47.3 years. The average age of discharges was 43.1 years and deaths 52.9 years.

For the year ending June 30, 1950, of those remaining in hospital diagnosed as manic-depressive psychoses, 32.8% of the first and 15.4% of the readmissions had been on the records 20 years or longer. This is again strongly suggestive that early intensive treatment would prevent many from becoming chronic cases and eventually requiring not much more than custodial care.

Even though the manic-depressive problem has been a decreasing one, especially for the past 10 years, they require an intensive treatment before release. The recovery rate is high, but the possibility of recurrence of this type of mental disorder is also high when the stress becomes great.

From past records the economic situation has a large part to play in the rate of admissions of manic-depressive psychoses. Therefore, it is difficult to show the trend to plan for hospitalization of this type of disease without also taking into consideration the economic trends. Of course, the increase in general population will always have some effect on the number of actual admissions even if the rate to population does not increase.

The Richmond-Henrico-Chesterfield Chapters of the American National Red Cross helped 6,361 servicemen, veterans or their dependents solve personal problems caused by their military service during the past year.

Free Red Cross home nursing or first aid training was completed by 4,726 Richmond area citizens in 1952. Civil defense officials urge at least one member of each family to become trained in first aid—a necessary skill in time of disaster.

More than 12,000 citizens of the Richmond area gave some of their time and skill as volunteers through the Richmond, Henrico or Chesterfield Red Cross Chapters, to help their fellow citizens during the past year.

Blood donors of Richmond-Henrico-Chesterfield Red Cross Chapters rolled up their sleeves and gave 7,315 pints of blood in the past year to help save the lives of many wounded servicemen in Korea.

A goal of $331,445 has been set for the Richmond-Henrico-Chesterfield Red Cross campaign March 5-24.

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Public Relations Conference Luncheon

THE MEDICAL SOCIETY OF VIRGINIA

Annual Public Relations Conference

The Annual Public Relations Conference of The Medical Society of Virginia was held on Thursday, January 29, 1953, at the Hotel Roanoke. A gathering of fifty, including representatives of fifteen component societies, was on hand to participate in the work-shop type program. Six PR projects were offered for the consideration of those attending, with the hope that they might be sponsored by the component societies.

Dr. James L. Hamner, President of the Society, opened the Conference with a word of welcome, and then introduced the presiding officer, Dr. John T. T. Hundley.

In presenting the 1953 Public Relations program of The Medical Society of Virginia, Dr. James P. King, PR Chairman, stated that the medical profession is becoming increasingly PR minded and no longer is on the defensive. Dr. King went on to outline the objectives for the year, and placed special emphasis upon the six projects on the Conference program.

The success story of the West Virginia State Medical Association in the field of Press-Radio Relations was told by Dr. Frank J. Holroyd, member of the West Virginia Council, and a pioneer in PR work in that state. Dr. Holroyd pointed out that things were not always so good in West Virginia, and the necessity of getting the physician's story told brought about dinner meetings with representatives of the press and radio.

The result was the adoption of a code of ethics and cooperation, which was approved by the State Press Association and the State Radio Association. The code listed the types of information available, treated editorial judgment, and arranged for medical societies and hospitals to designate spokesmen.

The part good public speaking plays in public relations was explained by Mr. Harry Green, President, Phillips Business College, Lynchburg. Mr. Green stated that there exists today no force which can compare with the live, human force. The impact of a message delivered personally is tremendous.

According to Mr. Green, some large corporations are spending millions training their employees in the art of speaking. The trend today is for as many people as possible to carry a message to the public —not leaving the task to just one or two. The better the participation—the better the public relations.

The value of sponsoring a health exhibit was emphasized by Mrs. Maynard R. Emlaw and Mrs. Hawes Campbell, members of the Woman's Auxiliary of the Richmond Academy of Medicine. Mrs. Emlaw described many of the exhibits available from A.M.A. and explained that the only real expense was the payment of shipping charges. Order blanks, she said, may be secured from the state office of The Medical Society of Virginia.

Mrs. Campbell described the work of the Auxiliary in making possible an obesity exhibit at the 1952 Atlantic Rural Exposition. She pointed out that such an exhibit is a wonderful project for the Auxiliary and explained how the work of the Auxiliary members made it a real success. Mrs. Campbell stressed the fact that besides being most worthwhile, sponsoring an exhibit can also be a lot of fun!

The Conference luncheon featured a talk by Mr. W. H. Radebaugh, PR Adviser, Textile Fibers Dept., E. I. du Pont de Nemours & Co., Wilmington, Delaware, concerning "The Importance of Public Relations." Mr. Radebaugh told of how the activities of very small groups had resulted in decisions of national importance, and he spoke strongly for sound, active PR programs on the local level.

A demonstration of the PR Course for Office Personnel, now being offered by the Public Relations Committee of the State Society, opened the afternoon session. Highlighting the demonstration was a playlet featuring Miss Marie Farris and Miss Helen Pugh of Lynchburg. The playlet depicted the right and wrong ways of talking with patients on the telephone, and emphasized the important role played by the physician's secretary in the conduct of his office activities.

Any component society can arrange to have the course presented in their locale simply by contacting the State Office. It was brought out that arranging

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Mrs. Hawes Campbell explains the value of health exhibits. Others are (l to r) Dr. John T. T. Hundley, Mrs. Maynard R. Emlaw, Dr. Frank J. Holroyd, and Mr. Harry G. Green.

Talking It Over

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for the course is an excellent project for the Woman's Auxiliary. The course is presented in two evenings, and involves no expense to those attending.

Tips on sponsoring a radio series were given by Mr. Alden Aaroe, well known announcer of Richmond station WRVA. Mr. Aaroe stressed the fact that there are over 105 million radio sets in use at the present time, and that radio will always flourish.

Radio stations, according to Mr. Aaroe, are always looking for good programs with listener appeal, but it must also be remembered that radio is a business and must show a profit. Therefore, anything presented as public service (time donated by station) must be especially good.

Mr. Aaroe mentioned the importance of programs directed to our youth, and recommended the "Dr. Tim, Detective" series for this purpose. Such a series is excellent when given good promotion and publicity. In this connection, he listed posters, newspaper notices, stuffers in statements, talks before school groups, etc.

The importance of "Working with Other Groups" was considered by the audience, which divided into three groups under Mr. M. A. Hubbard, Virginia Farm Bureau, Mr. J. Robert Anderson, State Department of Health, and Mr. Earl J. Shiflet, State Grange.

Each group had similar suggestions regarding the best way to work and cooperate with other groups, although Mr. Shiflet's report was accepted as the most practical. It was recommended that each group or profession first focus its attention on its particular problem before approaching other groups for assistance. Then, have a repesentative meet with representatives of the other group (or groups) and talk the matter over. Once a well thought out plan of presentation is agreed upon, the matter can be put before the public in the form of an educational program, etc.

In his summary, Dr. Holroyd had but one criticism of the PR program in Virginia, and that was the failure of the Society to make maximum use of the Auxiliary, which undoubtedly is one of the very best public relations mediums.

A CHECK LIST FOR YOUR PR WORKSHOP

(HOW MANY WILL YOUR SOCIETY CHECK IN '53)

	CHECK HERE
1. *Work for better press-radio relationship*	☐
2. *Organize a speaker's bureau*	☐
3. *Sponsor a health exhibit*	☐
4. *Sponsor a PR course for office personnel*	☐
5. *Sponsor a radio series for better PR ("Dr. Tim, Detective")*	☐
6. *Work with other groups*	☐

WHEN WRITING YOUR CONGRESSMAN, OBSERVE THESE TEN GOOD TIPS

FROM THE CHAMBER OF COMMERCE

1. Address him as Representative John Doe, or Senator John Doe.
2. Be Local; tell him how a National question affects *your* profession, *your* community.
3. Be Business-like; brief but not terse.
4. Be Specific; if you're *for* something, say so. If not, don't hedge.
5. Be Polite; Members of Congress deserve dignified treatment.
6. Re Reasonable; seek only possible things.
7. Be Yourself; use your own letterhead and letter style.
8. Request Action; your man is elected to *do* something.
9. Ask For An Answer; you've told him where you stand, now ask him where *he* stands.
10. Be Appreciative; thank him for good votes, compliment his better speeches, and praise his staff, too.

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MISCELLANEOUS

Attention, General Practitioners and Internists

On April 9, 1953, there will be held in Washington, D. C., a conference of the Metropolitan Washington Tuberculosis Association ("Metropolitan Washington" is comprised of the District itself and contiguous counties in Maryland and Virginia). Discussion will center on "The Role of the Private Physician in Tuberculosis," a topic much neglected in most official and voluntary control programs.

The program is sponsored by the Virginia Chapter, Academy of General Practitioners. Attendance of general practitioners in all three metropolitan areas is urged by current and past presidents of the Virginia Academy, as well as by the president of The Medical Society of Virginia. An excellent opportunity will be afforded the profession to further strengthen good public relations, albeit also an occasion "to talk and be talked about" with reference to yet another facet of its responsibility in the community, a challenge physicians can ill afford to ignore these days and times. Following an introductory address, conferees will temporarily disperse into smaller selected groups (one composed of physicians *only*) to permit uninhibited expression of opinion. Conclusions reached by each session will later be summarized and submitted to the conference as a whole for general discussion.

The opening paper of the conference will be presented by Dr. William F. Wagner, Director of the Division of Tuberculosis Control, Virginia State Health Department, who will elaborate briefly upon the following contentions as a focal point for discussion.

10,000 active cases are thought to exist in Virginia alone, most of whom await identification in a presently comparatively innocuous stage. As it becomes generally known that diagnosis of active tuberculosis no longer implies categorical need for hospital care, either for the patient's sake or to protect others, the public will be even more eager to participate en masse in community x-ray surveys. Many private physicians will be called upon to help meet this community problem. They can't all be "consultants".

Completion of diagnostic studies for tuberculosis is neither mysterious nor complicated when carried out according to protocol.

Repeated visits to a doctor's office may be required for months—but this is no more than is regularly accorded the prenatal case, as her due!

It is no more necessary for a general practitioner to be able to interpret a chest x-ray with any degree of finality than it is for a chest specialist to personally perform his own sputum examinations, if he has a certified laboratory to do it for him.

It takes less skill to do a tuberculin test or collect a gastric lavage specimen than it does to deliver a baby.

By and large, treatment of run-of-the-mill tuberculosis is simple—the real problem is treating the people who have it, their friends and relatives. The Health Department can help in these fields.

Specific drug therapy must not be looked upon as a substitute for sanatorium care, where the latter is indicated. Unless a physician is prepared, *as of now*, to treat tuberculosis *without* the newer drugs, he should not attempt to treat tuberculosis by simple resort to them.

Some general practitioners will be surprised, it is hoped all will be pleased, to learn that not only diagnosis, but treatment of the average case of pulmonary tuberculosis, *as found today*, requires no more, *if as much*, training and skill, as they must apply daily in their management of many other types of disease.

There should be no class of patients who will pay better, take less of the doctor's time, give him greater personal and professional satisfaction, than should the average tuberculosis patient of the next 10 years, who will appreciate *relatively* inexpensive medical supervision by his own family physician whose function it is to make as nearly unlikely as possible his ever having to quit his job to go to a sanatorium.

Now, as never before, is the time for *all* physicians, particularly those whose practices are not *strictly* limited to fields other than chest, to enlist personally and professionally in this potential crusade for improved community health; equally important, physicians should, through regular channels, prepare to solicit real support from appropriating bodies who serve localities, and state, in order that

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their own efforts may be implemented by such facilities and services as may be required to do a job, locally, and be supplemented by sufficient sanatorium beds to care for all in need of same.

Request for Reprints Concerning Stress and the Adaptive Hormones.

From current literature, we note that an ever increasing number of its articles deals with problems pertaining to research on "stress" and the so-called "adaptive hormones" (ACTH, STH, corticoids, adrenergic substances, etc.). In our opinion, the success of research in this complex and rapidly developing field largely depends upon the prompt availability and evaluation of relevant publications, a task for which we should like to solicit the assistance of your readers.

In 1950, our Institute has initiated the publication of a series of reference volumes entitled "Annual Reports on Stress" (Acta Medical Publishers, Montreal) in which the entire current world literature is surveyed every year (usually between 2000 and 4000 publications). Up to now, we had to compile the pertinent literature partly from medical periodicals, monographs, abstract journals and partly from reprints sent to us by the authors themselves. Of all these, reprints proved to be the best source of data which we felt deserved prompt attention in our annual reports. Hence, in the past, we have sent out several thousand individual reprint requests to authors of whom we knew that they are currently engaged in research on stress and allied topics. Even this procedure did not give us the wide coverage which would be desirable, because it is materially impossible to contact all these authors individually and it often takes too much time to get the requested reprints.

It is evident that in order to insure prompt inclusion of publications in the annual reports, these surveys must develop into a cooperative effort between the authors of original papers and the reviewers. This cooperation was greatly enhanced of late by the publication of announcements, in several medical journals, encouraging investigators interested in Stress Research to send us their reprints for this purpose as soon as they become available.

We should be grateful if your readers would advise of publication of articles on this subject to Hans Selye, M.D., Ph.D., D.Sc., F.R.S.(C), Professor and Director of the Institute of Experimental Medicine and Surgery, Montreal, Canada.

BOOKS

Thomas Jefferson: Scientist by EDWIN T. MARTIN. New York. Henry Schuman. 289 p. illustrated. Price $4.00.

In the references in the back of the book the author states that in this book he has used the term *science* to mean *all knowledge*. It is regrettable that this statement was not made in the first chapter so that the reader would read the book with a different standard. As it is, he gets a poor opinion of the science of the time and of Jefferson as a scientist. As a matter of fact, he was an encyclopedist rather than a scientist. All knowledge was his province rather than one particular field. His Notes on the State of Virginia, which is cited as a great scientific work, would scarcely be considered a book of science by today's standards. His interests ranged from paleontology to modern agriculture. He described and measured accurately a fossil with a claw 6½ inches long but diagnosed it incorrectly. He improved the plow and was awarded a gold medal from a French agricultural Society.

The most fascinating part of the work is the controversy between Jefferson and European scientists such as Buffon, DePauw, and Raynal over their errors about the effect of American climate on the fauna, especially the number of species and their size. Some 60 pages are taken up with this discussion.

M.P.R.

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The Toxemias of Pregnancy. By WILLIAM J. DIECKMANN, S.B., M.D., Mary Campau Ryerson, Professor and Chairman of the Department of Obstetrics and Gynecology of the University of Chicago; Chief of Service of The Chicago Lying-in Hospital and Dispensary, etc. Second Edition. St. Louis, The C. V. Mosby Company. 1952. 710 pages with 85 text illustrations and 1 color plate. Cloth. Price $14.50.

This book is born of the author's knowledge and research on the toxemias of pregnancy. As he reviews the literature having to do with the physiology and pharmacodynamics of pregnancy in their relationship to preeclampsia, eclampsia, nephritis, and the edemas, his comments flow out of a wide clinical and laboratory experience.

It was to be hoped that the interval between his first edition in 1940 and this one would see the discovery of the etiology of the toxemias. Unfortunately such is not the case. This edition actually contains little fundamental knowledge not previously presented. However, it serves a most useful purpose if for no other reason than to review the literature on the toxemias of pregnancy published during the last ten years and to present a critical appraisal of it in the light of the author's personal experience.

The organization and arrangement of material of this edition is superior to that of the first one. The physico-chemical determinations in pregnancy are succinctly covered in 75 pages of Chapter IV. Here are discussed all of the important biological and biochemical changes in blood chemistry, blood and plasma volume, hemoconcentration and hemodilution, hemoglobin and hematocrit during pregnancy. Blood oxygen saturation, leukocytes, blood platelets and blood coagulation, the serum and plasma proteins, and the colloid osmatic and oncotic pressures, fibrinogen metabolism, the acid base balance, and the diffusible and nondiffusible ions, venous and tissue pressures, and finally the complex changes in body water are reviewed.

The chapter on obstetric treatment of eclampsia is one which should be close at hand to everyone practising obstetrics. It is an excellent clinical outline of proper management of eclampsia and the toxemias reduced to the simplest form without sacrifice of essentials. The various complications of eclampsia and preeclampsia, such as pulmonary edema, vascular collapse, and uremia, are presented in the most scholarly and practical manner.

The only criticism that might be leveled is the author's presumption that all of his readers are prepared to understand such statements as "the calculated intracellular water compartment as the difference between the total body water and the bromide space", or "he noticed that the rhythmic gradient was markedly flattened by an increase in the rate of the ileum". Occasionally a loose statement appears which might be challenged, such as "the avoidance of normal work during pregnancy is thus desirable as a means of preventing overwork of the heart".

Everyone who practices obstetrics should have this "toxemia bible". For the laboratory worker and clinician interested in research the book is invaluable as a reference source. The bibliography is exhaustive. W. B.

The Human Pelvis. By CARL C. FRANCIS, A.B., M.D., Assistant Professor of Anatomy, Department of Anatomy Western Reserve University, Cleveland, Ohio. St. Louis, The C. V. Mosby Company. 1952. 210 pages with 61 illustrations, including 3 in color. Cloth. Price $5.00.

If the other chapters are as reliable and comprehensive as Chapters 1, 3, 5, and 7 (and there is no reason to doubt that an author of the standing and experience of Dr. Francis would be consistently meticulous and thorough throughout) this book, *The Human Pelvis*, should be a welcome addition to the orthopedic literature. The author's knowledge of the subject and of the research done in the field is extensive. His experience as a teacher and a researcher is shown by the clarity of description and the coherence of individual sections of the book and their relationship to each other. The illustrations are excellent and complete and it is a pleasure to report ability to read the identification of the structures shown. In the table presenting the segments of the cord which innervate the striated muscles of the pelvic region the author is not in complete accord with some of the anatomists and neuroanatomists. However, this need not be considered a serious deviation since there are wide variations in the combinations of spinal nerve fibers within the plexuses and the muscle fibers which they innervate.

The book as a whole, both in arrangement and in format, is excellent. E. D.

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EDITORIAL

William Baynham (1749-1814)

JUST above Loretto in Essex County, almost opposite the fork in the road to Hustle, stood the home of Dr. William Baynham, a country surgeon who, next to Philip Syng Physick, did more for the advancement of surgery than any one in the early history of America. He probably had the best medical education of anyone of his time.

William Baynham was the son of Dr. John Baynham of Essex County. His schooling began at Donald Robertson's famous boarding school on the Mattaponi River in King and Queen County. Among his schoolmates were James Madison, afterwards President of the U.S., Johnny Taylor, Edmund Pendleton "Jr.", Henry and James Innes, John Penn, two Lomax boys, and a son of John Hoomes. He studied medicine under his father and was apprenticed to Dr. Thomas Walker for five years. He then went to England for a stay of sixteen years.

In 1769 at the age of twenty, he entered St. Thomas' Hospital as a student. Here he became particularly interested in anatomy and won the respect and friendship of the professor, Joseph Else. In 1772 he was employed as prosector at Cambridge University and at the same time he practiced surgery in Margate in partnership with Mr. Slater. Concerning his work in the University, Charles Collignon, wrote Mr. Else as follows: "Sir—I beg leave to return you thanks for being instrumental in prevailing on Mr. Baynham to assist me, as his services have been entirely satisfactory, and his private behaviour very amiable and engaging. I am, sir, your very humble servant, Charles Collignon, Professor of Anatomy in the University of Cambridge." It was not long before Mr. Else wanted the young anatomist back in London with him. He wrote to Mr. Baynham: "Dear Sir—I am not well satisfied with Mr. Cline's manner of managing the dissections, and as he either will not, or cannot, make any considerable number of preparations—and as he is just married, I apprehend he will be less assiduous in future. There are several who are very expert in dissections, and would be willingly employed in his office, but there is none of whom I have so good an opinion as you. If, therefore, it could be contrived that you could be with me in the winter, for this purpose, to our mutual advantage, I should be glad. Hearing that you are shortly to be in town, I write to you, that if this matter is palatable to you, you may revolve it in your mind. I am, Sir, your most obedient servant. Joseph Else."

Arrangements were completed between the two which were very flattering to Baynham. "He was to superintend the anatomical theatre and dissecting room, prepare the bodies for his public demonstrations, make preparations for the museum, and to instruct his pupils in the art of dissecting, injecting, making anatomical preparations, &, with a salary of eighty and ninety pounds the first two years, and one hundred pounds a year for five succeeding years—at the expiration of which (having qualified himself in the interim for the office) Mr. Else was to relinquish to him the professor's chair, or to take him as joint professor, on equal terms, as he (Mr. Baynham) might choose." Unfortunately Mr. Else died of apoplexy five years later without making his plans known to the Board of Governors. In the election that followed Mr. Baynham lost to Mr. Cline by one vote. The museum which had been left him by Mr. Else he sold to Mr. Cline for 800 pounds. Some unfinished specimens in the museum he sold to Mr. Blizard, professor of anatomy at the London Hospital, for 100 guineas.

Baynham was regarded as the most skillful anatomist in Great Britain. He had

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mastered the technic of fine injection of blood vessels. His injection of the female breast was for a great many years regarded as a masterpiece. He discovered the rete mucosum and with his injection method settled many clinical problems. When shown one of Baynham's injection specimens Dr. William Hunter, who had been his teacher, is said to have remarked, "What have we been doing so long that we have never observed this before?"

The failure to succeed Mr. Else upset Baynham's plans. The anatomist now turned surgeon and on June 7, 1781 he became a member of the Royal College of Surgeons. For the next four years he practiced on equal terms with Pott, Abernathy, and John Hunter. Why he decided to return home and become a country surgeon is not explained by Anderson, Thacher or Slaughter. Blanton suggests that it may have been due to the ill feeling towards Americans after the British surrender at Yorktown in 1781, but there is no evidence of such animosity against William Baynham. If that were a factor it was a long time in taking effect. The irresistible lure of Virginia is a more likely reason, or it may have been that he had become tired of a big city. Such things do happen. Some years ago a young obstetrician came to see us about a location in which to practice in the South. He had just finished a residency in the largest lying-in hospital in New York City and he brought with him excellent letters of recommendation. In addition he had been offered a partnership with an excellent obstetrician who was known to have the most lucrative private practice in the metropolis. When we showed surprise that he did not take such an offer, he replied that there was not enough money in the world to make him stay another year in New York.

So in 1785, when he was thirty-six years of age, William Baynham returned to the land of his birth. His ability was quickly recognized and patients came from far and near. He performed all the operations of his day—cataract, stone and ectopic pregnancy. His two most famous ones were reported in the second number of the first volume of the *Medical and Philosophical Journal and Review*, "An Account of two cases of Extra-Uterine Conception; in each of which the Fetus was extracted by an operation with success." These cases give such a good picture of eighteenth century surgery that we give the second case in Dr. Baynham's own words. He attributes his success to his keeping the operation entirely within the gestation sac.

"On the 24th day of January, 1799, I was sent for by Mrs. Washington, of Hayfield, in the county of Fairfax, Virginia, to a servant of her's, a young woman who had been with child, as she asserted, eighteen months; she had heard that I delivered a woman who had been with child for ten years, and was willing to submit to any operation that I might think necessary for her relief.

"She was taken in labour last March, as she thought; the midwife was sent for; but after being a few hours in great pain, the pains left her; she was at the same time taken with the menses, which continued with her for nine weeks, when they left her, and she has had them regularly every month until the last period. She grew less every day, yet kept tolerably well, though oftentimes in a great deal of pain. About six weeks ago she was taken worse than usual, and has been wearing away ever since, having no stomach to eat any thing, always complaining of so heavy a load at the bottom of her belly, that when she stands up she is obliged to hold it up with her hands. I found her labouring under a hectic fever; she was much emaciated, and confined to her bed.

"The abdominal tumour was equal and uniform, reaching half way from the umbilicus to the scrobiculus cordis, but more prominent in the umbilical region, where the head

of the fœtus was plainly to be distinguished by the hardness of the feel.

"I performed the operation on the 6th of February, 1799. I carefully made an incision through the integuments and muscles in the most prominent part of the tumor, thereby exposing the head of the child, and cautiously extended it upwards and downwards by the direction of the finger introduced into the cavity, so as to reach from a little above the umbilicus nearly to the pubes, on the left side, close to and in the direction of the linea alba; by which a sufficient opening was made to extract the child with ease. It was of the common size, and sound, except on the head, where a small portion of the scalp was in an incipient state of putrefaction. A large branch of the internal mammary artery was divided; but it was readily secured by the needle and ligature. No liquor amnii or other fluid was found in the cavity, which closely embraced the fœtus, and to which it was attached and glued, as it were, by a sort of cement of a greyish cast, of the nature of that kind of mucus which is found on the surface of the bodies of new-born infants; so that I was obliged to insinuate my hand, and with my fingers carefully separate the head, body, and arms from their attachments. The funis was small and short, connecting the child with the mother by three or four ramifications, inserted low down in the anterior part of the cavity in which it was lodged. The adhesion was so strong that I did not think it advisable to use any force to separate it; choosing rather to leave it to nature to dislodge it. Some pieces were thrown off at almost every dressing; but it was upwards of a fortnight before the string came away; some days after which I left the patient to the management of the family, as the wound was by that time healing, the cavity filling up and closing fast. The same dressings were used in this case as in the first. An anodyne was given immediately after the operation, repeated again that evening, and for three or four nights afterwards; it was then discontinued, and the Peruvian bark given three or four times a day. The bowels were carefully kept open from the second day after the operation, first by emollient injections, afterwards by the occasional use of an infusion of senna, manna, and salts. The diet was at first light and slender, but afterwards changed for one more generous and liberal. She soon got perfectly well, and is now alive and in good health. I believe she has not been again pregnant."

Dr. Baynham was a man of excellent judgment. A contemporary described him as being "not a ready man but a full and accurate one." His enunciation was indistinct and he spoke slowly. He seldom made a favorable impression on strangers. He had formed very rigid notions with respect to almost every social obligation and was scrupulously exact in adhering to them. Although his exterior was gloomy and austere, his heart was warm and generous. In him the poor ever found a friend and benefactor ready by the humane offices of his skill to assuage the pangs of bodily disease or by his beneficient liberality, which sometimes bordered even on the munificent, to relieve them from the equal pangs of poverty.

Little is known of the Baynham family in Mid-Tidewater, Virginia, today. It seems to have completely died out. Edmund Pendleton mentions Baynham in his will "to my physician five guineas to buy a rug or such other memorial."

Dr. Baynham married a daughter of the Rev. John Mathews. Their son, William Armistead Baynham, graduated in medicine but practiced only a few months. He became a celebrated Baptist preacher and did much good with his inherited fortune, besides giving Enon Church in Essex County its brick building.

Nothing remains to remind us of this great XIX century Virginia surgeon. One wonders if any of the magnificent anatomical preparations he made for London museums are still in existence.

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SOCIETIES

The Alexandria Medical Society

Was host at a joint meeting of the Arlington, Fairfax and Alexandria Societies in Carter Hall, Alexandria Hospital, on January 15, at 8 p.m. Many problems of local interest were discussed. It was proposed and approved by all present that a joint committee be set up as a 10th Medical District coordinating group, for the purpose of liaison in handling conditions of interest to the entire area.

For several years the Alexandria Society has held an annual Clinic Day in the Spring. This year it will be sponsored jointly with the other societies in the 10th District. Many able speakers are invited and much worthwhile material is presented. It is hoped to have a large attendance.

Fourth District Medical Society.

At the annual meeting of this Society, Dr. Edith Miller of Petersburg was elected president; Dr. H. B. Showalter, Kenbridge, and Dr. Lee Scott Barksdale, Hopewell (Dr. Barksdale has since died) as vice-presidents; Dr. James A. Thweatt of Petersburg was re-elected secretary-treasurer; and Dr. Tyree Finch of Farmville was named chairman of the Steering Committee. Several counties turned in names of their delegates and alternates to The State Society meeting to be in Roanoke.

The Society has a Grievance Committee of the Petersburg Medical Faculty and is in process of setting up an Emergency Call Service in Petersburg.

The James River Medical Society

Held the election of officers at a meeting on January 14 at the Hotel William Frank at Fork Union. Those elected are: President, Dr. William S. Lloyd, Goochland; vice-president, Dr. A. C. Whitley, Palmyra; and secretary-treasurer, Dr. J. H. Yeatman, Fork Union.

Albemarle County Medical Society.

At the meeting of this Society on January 8, Dr. E. D. Davis of Crozet was elected president for the current year and Dr. Armistead P. Booker was re-elected secretary-treasurer. Dr. Byrd S. Leavell of the University of Virginia was named vice-president. Dr. J. G. Bruce, Jr., of Gordonsville, and the president, Dr. Davis, were named as representatives to attend the Rural Health Conference, sponsored by the Council on Rural Health of the American Medical Association, to be held in Roanoke, February 27-28. Dr. Vincent W. Archer of the University and Dr. Thomas S. Edwards of Charlottesville were named as the Society's representatives to the annual Public Relations Conference of the State Society to be held at Hotel Roanoke, January 29.

Warwick-Newport News Medical Society.

At the regular monthly meeting of this society at the Warwick Hotel, Newport News, on January 13, the speaker was Dr. Ed. Levy, clinical pathologist at the Norfolk General Hospital. His topic was "Recent Advances in Diagnosis and Management of the Blood Dyscrasias".

Dr. O. Watts Booth is president of this society, and Dr. O. Ward Anderson, Jr., secretary-treasurer.

Lynchburg Academy of Medicine.

At the regular monthly meeting of the Academy on January 12, at the Lynchburg General Hospital, Dr. J. H. Allan, professor of Orthopedics at the University of Virginia was the guest speaker, his subject being "Cervico-brachial Pain." Dr. P. G. Hundley was elected to honorary membership. The secretary named for 1953 is Dr. Frank N. Buck, and assistant secretary is Dr. Richard Hawkins. Both are of Lynchburg.

Richmond Academy of Medicine.

At the meeting on February 10, the Section on the History of Medicine was host to the general membership of the Academy, and the members and their wives were guests. The program included a paper on "Osler and Welch—Founders of Modern American Public Health" by Dr. Huntington Williams, Commissioner of Health from Baltimore, and "The Exodus of Southern Medical Students from Philadelphia in December 1859" by Dr. Thomas W. Murrell, Sr., of Richmond.

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NEWS

Dr. H. C. Rucker, Virginia's Representative.

Dr. H. Cowles Rucker of Mattoax has been appointed by Governor Battle as Virginia's guest of honor at the Western Hemisphere Conference to be held in conjunction with the World Medical Association in Richmond, April 23-25. This meeting is to mark the diamond anniversary of the progress of medicine, and each State has been invited to designate some doctor now in practice who is approximately seventy-five years of age. Dr. Rucker will be just seventy-five and was several years ago honored by the people in his section for his services in that community.

Recently Governor Battle asked each of his fellow-Governors to appoint a physician who will reach the age of 75 during 1953 to visit Virginia for this meeting and tell of medical advances that have taken place during his lifetime. The invitation included the physician's wife.

At the Richmond conference, guests will be greeted by Dr. Louis H. Bauer, president of the American Medical Association, and by leaders of Latin American medical societies. Besides scientific sessions, there will be opportunities for visiting historic sites in Virginia, including the 18th century Williamsburg restoration. Expenses of the conference, and of guests and delegates, are covered through a grant by A. H. Robins Co., Inc., ethical pharmaceutical house founded in Richmond 75 years ago this year.

With the appointment of Dr. Rucker, 36 Governors have designated guests of honor to the coming conference.

Some Objectives for 1953

As recommended by Dr. Louis A. Bauer, President, A.M.A. are:

1. Work with rural communities to establish facilities for physicians, so that we shall have a better distribution of physicians.
2. See that good medical care for the indigent is available everywhere just as it is in some states.
3. Extend public health coverage to areas lacking it.
4. Develop plans for the care of the chronic invalid.
5. Expand our voluntary insurance program, not only to cover more persons, but to cover those over age 65 and those suffering from illness of long duration.
6. Clean our own house, by disciplining those physicians who are tarnishing the reputation of the whole profession by their unethical acts of overcharging, accepting kick-backs, and making commercial arrangements with pharmacists.
7. See that the public is protected so that they can always obtain the services of physicians.
8. Revitalize our county societies and make them leaders in their communities in all health matters.
9. Inculcate the newly trained physicians in the tradition and ethics of medicine.

The Virginia Radiological Society

Will hold its mid-year meeting on Friday and Saturday, April 24 and 25 at the Hotel Roanoke, Roanoke, Virginia. The meeting will begin with a film interpretation session Friday at 8 p.m. and will continue through Saturday afternoon. The feature of the meeting Saturday will be an address by Dr. Ernest H. Wood, Professor of Radiology, University of North Carolina, at a luncheon meeting. There will be a dinner Saturday evening.

TB Symposium for GP's in Saranac Lake Next Summer.

The Second Annual Tuberculosis Symposium for General Practitioners will be held in Saranac Lake, New York from July 13 through 17, 1953. It is approved by the American Academy of General Practice for 26 hours of formal credit for its members. The Symposium is sponsored by the Saranac Lake Medical Society and the Adirondack Counties Chapter of the New York State Academy of General Practice. The registration fee is $40 for A. A. G. P. members and $50 for non-members. Registration is limited to 100 doctors.

Many physicians who attended last year's symposium brought their families to Saranac Lake. So that families might have use of the car to enjoy the many recreational facilities of the Adirondack Mountains, free bus transportation was provided for physicians from Saranac Lake to the various meeting places. This practice will be followed again this

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year. The 1953 Symposium has been planned to cover all important aspects of pulmonary tuberculosis from the General Practitioner's point of view.

Many of the sessions are informal panel discussions with ample opportunities for questions from the audience.

The Symposium will be held in various sanatoria and laboratories in the Saranac Lake area. On Monday, July 13th there will be a dinner for physicians attending the course, their families and the faculty. The speakers and panel members at the Tuberculosis Symposium will include physicians, surgeons and scientists from Saranac Lake and surrounding areas.

Complete information concerning this program can be obtained by writing: Richard P. Bellaire, M.D., Tuberculosis Symposium for General Practitioners, P. O. Box 707, Saranac Lake, New York.

"The Role of the Private Physician in Tuberculosis".

This topic is to be discussed on April 9, at a conference to be held in Washington. The program is prepared especially for doctors in the District of Columbia and adjoining counties in Virginia and Maryland. Attention is called to the program elaborated on page 178, in the article "Attention, General Practitioners and Internists".

Spring Congress in Ophthalmology, Otology and Allied Specialties.

The Gill Memorial Hospital of Roanoke announces its twenty-sixth Annual Spring Congress in Ophthalmology, Otology, Rhinology, Laryngology, Facio-maxillary Surgery, Bronchoscopy and Esophagoscopy to be held in that city April 6 to 11. The lectures will be given at the Patrick Henry Hotel and the operations and clinical demonstrations will be at the Gill Memorial Hospital. The faculty includes between twenty and twenty-five specialists of prominence from various parts of the United States, with one doctor from London, in addition to members of the hospital staff. There will be a number of commercial exhibits. Entertainment and trips to points of interest are to be enjoyed by the doctors and ladies accompanying them.

Appointments to Medical Services in Petersburg Hospital.

Eight physicians have been appointed chiefs of medical services in the Petersburg (Va.) General Hospital. These were appointed according to regulations of a joint commission for accreditation of hospitals and each chief will be responsible for his particular service. Those appointed are:

Dr. William S. Grizzard, obstetrics
Dr. Nelson M. Smith, general surgery
Dr. Wm. B. McIlwaine, III, pediatrics
Dr. Joseph P. Whittle, medicine
Dr. Meade Edmunds, surgical specialties
Dr. James H. Powell, general practice
Dr. Carroll A. Peabody, radiology
Dr. Joseph R. McCormick, pathology

The hospital was opened on January the 16th and has been practically filled ever since.

Medical Orientation Conferences, Second Army Area.

Following previous policy, the Medical Section, Second Army, is taking the Medical Orientation Conferences to the field so as not to infringe on the professional time of Reserve Medical Officers. They will be conducted on weekends with a starting time at 1:30 p.m. at

Cleveland	March 14
Cincinnati	March 15
Philadelphia	March 21
Pittsburgh	March 22
Richmond	March 28
Baltimore	March 29

Dr. Clyde H. Dougherty,

Hopewell, has been made medical advisor to the Hopewell Emergency Crew, succeeding Dr. Lee Scott Barksdale, deceased.

Dr. Forrest M. Swisher,

Of the Anderson Clinic, Arlington, has successfully passed the examination for certification by the American Board of Orthopedic Surgery and is now a diplomate of that Board.

Dr. S. G. Miller,

Formerly of Nelson County, Virginia, but who had practiced in Covington since 1940, has accepted a position on the staff of Grace Hospital, Banner Elk, N.C., and entered upon his duties there on February 1.

Dr. William Meyer,

Herndon, has been elected illustrious potentate of Kena Temple, Ancient Arabic Order of the Nobles

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of the Mystic Shrine of Northern Virginia, to serve for the year 1953.

Richmond Eye Hospital.

Dr. Rudolph C. Thomason has been re-elected president of the Board of Directors of this hospital. Other officers of the Board are Dr. B. Randolph Wellford and Dr. L. Benjamin Sheppard vice-presidents, and Mr. Nathan Bushnell, III as secretary-treasurer. Other Board members are: H. Harry Augustine, Dr. John Burke of Washington, George E. Haw, Dr. S. M. Cottrell, Dr. W. Wallace Gill, Dr. E. Trible Gatewood and Dr. Edwin D. Vaughan (new member). During the meeting the Board confirmed the elections of the Hospital's medical staff: Dr. Edwin Vaughan, chairman; Dr. Edgar Childrey, Jr., chairman-elect; Dr. Edmund LaPrade, recording secretary; and Mr. Bushnell corresponding secretary.

The American Congress of Physical Medicine and Rehabilitation

Will hold its annual scientific and clinical session August 31, September 1, 2, 3 and 4, 1953 inclusive, at the Palmer House, Chicago, Ill. All sessions will be open to members of the medical profession in good standing with the American Medical Association.

In addition to the scientific sessions, annual instruction seminars will be held. These lectures will be open to physicians as well as to therapists, who are registered with the American Registry of Physical Therapists or the American Occupational Therapy Association.

Full information may be obtained by writing to the executive offices, American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago 2, Illinois.

Mr. Don E. Gilliland,

Wyeth district sales manager of the St. Louis territory for the past five years, has been appointed division sales manager of that Company at Cleveland. He has been with Wyeth eight years, starting as a special representative in the biological and allergen promotion field.

The Postgraduate Course in Diseases of the Chest,

Sponsored by the American College of Chest Physicians, Pennsylvania Chapter and the Laennec Society of Philadelphia, will be presented at the Bellevue-Stratford Hotel, Philadelphia, March 23-27, 1953. This course will emphasize the recent developments in all aspects of the diagnosis and treatment of chest disease, and is open to all physicians; however, the number of registrants will be limited.

The tuition fee is $50 and applications will be accepted in the order in which they are received. This course has been approved for credits by the American Academy of General Practice. Applications should be sent to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Have You Contributed?

Referring to the National Fund for Medical Education, to which the American Medical Education Foundation of the A.M.A. is contributing, President Eisenhower, one of the founders of the Fund, recently said:

"Private support of higher education in America is fundamental to the democratic tradition. American business must assume a greater share of the operating costs of our institutions or they shall have no recourse but to Federal subsidy—a perilous alternative.

"The nation's 79 medical schools are the keystones of national health and prime factors in our ability to maintain production levels and man our armed services.

"They must not be prevented, because of financial stress, from meeting these responsibilities."

Remember these words, and get your contribution in to the American Medical Education Foundation.

Symposium on Stress.

The Division of Medical Sciences of the National Research Council and the Army Medical Service Graduate School, Walter Reed Army Medical Center, will sponsor jointly a Symposium on Stress on March 16-18, at the Sternberg Auditorium, of the Army Medical Service Graduate School, Washington, D.C.

The American Goiter Association

Will hold its 1953 meeting in the Drake Hotel, Chicago, May 7, 8 and 9. The program will consist of papers and discussions dealing with goiter and other diseases of the thyroid gland. Dr. Claude J. Hunt of Kansas City, Mo., is president. Other information may be obtained from Dr. George C. Shivers, corresponding secretary, 100 East Saint Vrain Street, Colorado Springs, Colorado.

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For Sale to Settle an Estate.

Mobile X-Ray Department. Sacrificed at $1,500.00 1—G.E. Model D X-Ray Unit like new; 1—14x17 Cassette good par speed screens; 1—8x10 Cassette good par speed screens; 1—Portable Buck; 1—Fluorescent Illuminator; 1—Kodak safe light; 1—3-gal. three compartment Developing tank, and interval timer.

Also 1—Scanlan-Morris Battery Sterilizer consisting of: Autoclave 12x24; Water Sterilizers (Hot and cold) 5-gal. capacity; Instrument & Utensil Sterilizer 20x24; Electrically operated on 230 v. Power. Good condition—approximately twelve to fifteen years old. $500.00. Write Mrs. R. C. Fravel, Woodstock, Va.

OBITUARIES

Dr. Lee Scott Barksdale,

Prominent physician of Hopewell, died January 28 in a Richmond Hospital. He was a native of Petersburg and forty years of age. He graduated in medicine from the Medical College of Virginia in 1937, later taking up post-graduate work. He served as health officer in Hopewell in 1939 and after a time entered private practice. Dr. Barksdale served in the Navy Medical Corps for three and a half years and was discharged with the rank of lieutenant-commander. He was a member of The Medical Society of Virginia and interested in medical affairs of his section. He is survived by his wife and a son.

Dr. John Gibson Davis,

Oldest practicing physician of Roanoke, died February 1, at the age of 89. He was a graduate of the Medical College of Virginia in the class of 1885 and was a member of the first class to take the State Board of Medical Examiners. After practicing in West Point and at Boones Mill, Dr. Davis located in Roanoke in 1905 where he had since made his home. He had five sons, a granddaughter, a grandson and a brother who were all graduates of the Medical College of Virginia. He had been a member of The Medical Society of Virginia since 1914.

Dr. William Walker Robertson,

Danville, believed to be the oldest physician in that section, died January 19, at a local hospital, where he had been a patient for three years. He graduated from the former College of Physicians and Surgeons of Baltimore in 1890 and practiced for a number of years in Pittsylvania County before locating in Danville in 1921, where he practiced for some years before retiring. He had been a member of The Medical Society of Virginia since 1891. A son, Dr. E. B. Robertson of Danville, survives him.

Dr. John Marion Love.

The following resolutions were presented at the regular business meeting of the Norfolk County Medical Society January 5, 1953:

Dr. John M. Love was born in Petersburg, Virginia, and died at his home in Norfolk, December 6, 1952, 74 years of age, following a short illness. After his graduation from Hampden-Sydney College and Johns Hopkins University he spent considerable time studying in the European Clinics and post graduate work in eye pathology at Harvard University. He began his practice in Norfolk and later volunteered his services in World War I and was sent overseas with the United States Army. After his discharge from the Army he continued the practice of his specialty with the Veterans' Administration until he retired a few years ago because of age requirements.

He was a member of the American Medical Association, The Medical Society of Virginia, the Norfolk County Medical Society and the Tidewater Ophthalmological and Otolaryngological Society.

He will be remembered by the members of the profession for his high sense of honor, integrity and ethical standing.

We ask that this memorial be inscribed in the minutes of the Society and a copy sent to the Virginia Medical Monthly and to the family.

(Signed) J. WARREN WHITE, *Chairman*
W. L. HARRIS,
F. O. FAY.

Dr. George Howard Salomonsky,

Retired Veterans Administration physician, died February 5 at a hospital in Roanoke. He was forty-nine years of age and a graduate from the Medical College of Virginia in 1931. He was chief of the professional section Roanoke regional office of the VA from March 1948 until his retirement last June. He was recently awarded the Citizens Salute of the Disabled American Veterans for his services to veterans. He is survived by his wife and two children.

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